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FIBROID DISEASES OF THE LUNG.

INCLUDING

FIBROID PHTHISIS.

BY

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WITH TABLES AND EIGHT PLATES IN COLOURS.

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PREFACE.

When this book was undertaken, we had for some time previously been engaged in collecting a very considerable amount of material bearing on Fibroid Disease of the Lung. The number of cases which we had met with during the space of three years, at the City of London Hospital for Diseases of the Chest, and at the London Hospital, had grown to such an extent, and many of them were such typical instances of the disease, that we had resolved to publish them.

About this period, Sir Andrew Clark was giving some clinical lectures at the London Hospital, and one of us, as his *chef de clinique*, brought under his notice a few of these cases. Sir Andrew himself was also at the time contemplating a work on the subject, and hearing of our intentions, with characteristic kindness proposed that we should produce the book together. It was therefore decided that we should jointly write the account of Fibroid Disease which is now placed before the profession.

When the undertaking was nearing its completion, Sir Andrew Clark's death took place; but before this event occurred, the whole of the work had been written, and twice revised by all engaged upon it. Since then no material alteration has been made, either in the plan of the book, or the views expressed therein.

It is well known that Sir Andrew Clark's many and important engagements fully occupied his time, and we feel that we owe a deep obligation to him for the ready way in which he devoted his leisure and his unique experience to the work; indeed, much of his last vacation was spent in the service of this volume.

Our first idea was to bring out a book of plates, with short descriptions illustrating the various morbid conditions found in fibroid lungs. Later, as more material came to hand, we decided

to enlarge the scope of the work, and to examine more thoroughly the whole subject of Fibroid Diseases of the Lungs. We have, therefore, collated the views to be found in most of the literature upon the subject, and have endeavoured to make the book useful for purposes of reference; based as it is upon an inquiry as searching as possible, and illustrated by a larger number of typical instances of the disease than, we venture to think, has ever before been brought together.

Our thanks are due to many who have aided us in the work. We would express our indebtedness to our colleagues at the City of London Hospital for Diseases of the Chest, for the kind way in which they have allowed us to make use of the cases under their care; and to Dr. Barlow for furnishing us with the excellent description of one of the cases figured in the Plates.

The very faithful drawings of the microscopic changes in the lung were made by our friend, Dr. H. G. Adamson; and to him, as well as to our friend Dr. Kington Fyffe, who undertook the laborious work of reading the proofs, and made many valuable suggestions, our best thanks are due.

W. J. H.
A. C.

LONDON,
September 1894.

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FIBROID DISEASES OF THE LUNG.

CHAPTER I.

HISTORICAL ACCOUNT.

Fibroid Disease of the Lung has been known and discussed under various names ever since the days of Bayle and Broussais, but though frequently described, there can hardly be found any two writers on this subject who agree as to the pathology and clinical aspects of the disease. Thus, Sutton termed it "Fibroid Degeneration," and maintained it to be a constitutional disease, induced by a special fibroid diathesis.* On the other hand, Corrigan before this had described another form of the disease under the name of "Cirrhosis," which he held was of local and not constitutional origin, and which anatomically had its chief seat in the interstitial tissue of the lung. Owing to this wide diversity of opinion, the nomenclature of the disease has been involved in much perplexity and confusion. Each writer, holding his own particular views as to its morbid anatomy and pathology, adopted that terminology which seemed to him to convey most accurately the main idea of his belief. Hence arises the multiplicity of names for these processes taking place in the lungs.

Some authors—*e.g.*, Laennec and Stokes—gave a fairly complete account of the disease, while describing another morbid condition which was closely associated with it. Both these authorities, in giving illustrative cases of dilatation of the bronchi, described fibroid conditions of the lungs, which they believed to be either secondary to the dilatation, or indeed, of accidental origin. Others, among whom may be mentioned Grisolle, Chomel, Charcot, Andral, and Wilson Fox, styled the affection "Chronic Pneumonia," notwithstanding the doubt existing in their minds as to

* Thus adopting the view promulgated by Handfield-Jones.

the chronic condition succeeding the acute. Some of the names were applied on account of the colour and texture of the lung when invaded by this process. For instance, Bayle called it "Melanosis"; Addison, "Grey Induration"; while Corrigan, supposing the disease to resemble the poly-lobular changes in the liver, gave to it the name of "Cirrhosis." Handfield-Jones discussed the whole question of a constitutional fibroid change taking place in the different tissues of the body, and applied to it the term "Fibroid Degeneration." Sutton also gave it the same name, and in common with Handfield-Jones regarded it essentially as a degenerative process, in which the lung substance proper was replaced by a tissue of lower formation. Iuergensen and Rokitansky held it to be really an interstitial inflammatory process, in other words, extra alveolar, and preferred to call it "Interstitial Pneumonia." Drs. Hughes Bennett and Wilson Fox maintained strongly the view that these processes were in reality tubercular, or the result of cured tubercle.

In 1868, Sir Andrew Clark drew attention to the subject in a paper read before the Clinical Society, in which, whilst asserting that when invading the lung and accompanied by cavities, it was a phthisis in the strict acceptation of the term, he acknowledged the main features of the change to be a fibrosis, and gave it the name of "Fibroid Phthisis." By this title the disease is now generally known in this country.

We have then for this disease the names of "Fibroid Phthisis," "Fibroid Degeneration," "Cirrhosis," "Chronic Pneumonia," "Interstitial Pneumonia," "Grey Induration," and "Melanosis."

The acceptance of the doctrine of Laennec, which, expressed in brief, regards every cheesy mass as tubercular, soon divided observers into two classes: (1) those who believed that the primary cause of this Fibroid Phthisis was tubercle, and (2) those who believed that although fibroid phthisis was in some degree almost invariably connected with tubercle, it might, and indeed, did have an origin quite independent of tubercle.

Literature.—In examining and reviewing the literature devoted to this subject, it must be borne in mind how far back in medical history some of the accounts are to be found, and the difficulties in the way of writers of those times. We must remember that the first of them had no stethoscope, that physical examination in his day was but little developed, and that *post-*

mortem observations were uncommon. Later on, we find the use of the stethoscope advancing the accuracy of the recognition of the physical signs noted, but as yet only naked-eye appearances of the *post-mortem* examination are recorded, for these early observers had no microscope. From the invention of the stethoscope and microscope up to the present time, we may notice the ebb and flow of opinion one way and another, gradually leading to the recognition of what we now believe to be the usual characteristic course of the disease—the signs and symptoms *before* death, associated with the no less characteristic conditions of the lungs, and other organs to be observed at the general and microscopical examinations *after* death.

Carefully bearing in mind, then, what we have in hand—viz., Fibroid Disease of the Lung with or without ulceration—and taking into consideration the various antecedent conditions which may give rise to this affection, we must study, under whatever designation we may find them classed, accounts of cases and pathological researches dealing with any of these conditions, which—although they may be different in their beginnings, and although, further, they may slightly differ in their course and *post-mortem* appearances—tend nevertheless ultimately to this affection.

We must be prepared, as stated, to find authors treating of the subject under different names, describing different pathological conditions, and holding diverse opinions as to its origin. We hope to be able from the mass of information thus gleaned, to arrive at a more correct idea of the various ways in which these conditions may arise, and of the different signs and symptoms attending their progress and termination.

Bayle.—The first writer touching on the subject is Bayle, who, in his *Recherches sur la Phthisie*, published in 1810, speaking of phthisis with melanosis, described six cases of induration of the lung, with contraction and deposits of black pigment. At about the same period, Broussais published accounts of chronic pneumonia; but Chomel, writing some years later, was inclined to think that Broussais had mistaken the results of pleurisy and phthisis for this condition. Bayle (after describing a tuberculous phthisis, in which tubercles became aggregated in masses, and eventually broke down; and a granular phthisis, in which the tubercles were hard, translucent, and showed no sign of caseation),

proceeded to describe yet another form of phthisis—the phthisis with melanosis, a condition in which the lung was hard, compact and black, and sometimes resembled cartilage in structure. Cavities were often seen to exist, and Bayle observed that both the tuberculous and granular forms of phthisis sometimes complicated the melanotic variety—this complication, in the case of the tuberculous variety, being attended with shortened life, in the case of the granular form influencing scarcely, if at all, the progress of the case.

Bayle says, that in a lung affected with melanosis, the pleura is generally thick and adherent to the chest wall, and that the bronchi are sometimes dilated. The disease often lasts a long time, and attacks, most frequently, people well advanced in years; indeed, the ages of four of his cases were respectively, 62, 69, 62 and 74. Chief among the symptoms of melanosis, according to this authority, are cough and expectoration. There is little or no hectic, and no emaciation. Towards the end of the case, œdema of the legs is apt to supervene. Bayle found the position of the induration and deposit to be subject to wide variation, sometimes being confined to the lower lobe, sometimes to the upper, and in one case the whole lung was more or less affected.

Whatever may be the real pathology of Bayle's six cases, he makes it clear that the predominating feature of the disease is a pigmented induration. In some of his cases, as has already been remarked, there was a total absence of tubercle, and these may possibly have been cases of pure fibroid induration. The other cases were associated with tubercles in some form or other. But as to whether the tubercles were primary or secondary, does not appear. However this may be, the following points are clear: 1st, the undoubtedly fibroid condition of the lungs described, and the frequent presence of cavities therein; 2nd, the occasional existence of tubercles; 3rd, the dilatations of the bronchial tubes, in some cases; 4th, the thickened and adherent condition of the pleura; and 5th, the long duration and chronic course of the affection, attended with much cough and expectoration, but with no emaciation or fever, and often accompanied by œdema in its last stages.

Laennec.—In 1819, that great French physician, Laennec, gave to the world his celebrated work on Mediate Auscultation. In it there is information concerning fibroid disease under the

head of dilatation of the bronchi, and again under that of chronic pneumonia. In speaking of bronchial dilatation, Laennec described the induration met with around the tubes. He regarded the dilatation as the primary affection, and the induration as secondary. He represented the upper lobes to be most commonly the seat of this disease, and noted the absence of tubercles. According to Laennec, bronchial dilatation is most common in children, yet of the four cases which he relates, only one is a child, the others being between the ages of 40 and 70. In two, if not three of these cases, there existed fibroid changes, the lung being much contracted, and the pleura thickened. Both lungs were more or less affected in two out of the three cases, and in one instance gangrenous cavities were found involving both lungs, with, in addition, patches of recent catarrhal pneumonia, which probably had been the immediate cause of death. Laennec's theory as to the production of dilatation, was that the tubes were temporarily distended by an accumulation of secretion, and that the dilatation thus produced was then rendered permanent by the continual secretion of sputum. As a result of this dilatation, the intervening lung-tissue became compressed and condensed.

Perhaps it will be well to give the important features of Laennec's four cases. The first was that of a child, who died of whooping-cough. At the autopsy, the bronchial tubes in the left lower lobe were dilated, and the intervening pulmonary tissue compact, though flabby. This, Laennec called acute dilatation of the bronchi. The second case was that of a woman, aged 72, who had suffered for upwards of fifty years from dyspnœa, expectoration, and occasional hæmoptysis. Latterly, she was attacked with diarrhœa and œdema of the legs. At the necropsy, dilatations of the bronchial tubes were found chiefly in the right superior lobe, and the lung-substance was firm and condensed. Case three, was that of a man who had complained of cough, dyspnœa and muco-purulent expectoration ever since an attack of pneumonia, twenty years before. The left side of the chest was one-third smaller than the right, and there was well-marked bronchophony around the lower angle of the scapula. The man died suddenly, with symptoms pointing to apoplexy. On examination, the left lung was reduced to the size of two fists, and was closely adherent to the pleura. The whole lung was converted

into a cartilage-like, fibrous material. The upper lobe was slate-grey in colour, and the lower as white as tendon. The bronchi were everywhere dilated. The fourth case was that of a man, aged 41, who had suffered from cough, expectoration and dyspnœa from infancy; latterly, he had fever, diarrhœa, night sweats, emaciation, and hæmoptysis. There was contraction of the lower part of the left chest, with bronchophony and mucous rhonchus. After death, the right lung was found to contain many areas of induration. The left lung was traversed by dilated bronchi, which were especially numerous in the lower lobes. The intervening pulmonary tissue was condensed, firm, and, in some parts, wholly cartilaginous, forming one mass with the "degenerated envelopes." Of these four cases of Laennec—the second, third, and fourth, are undoubtedly fibroid; indeed, the third case is as typical an example as can well be imagined of that form of fibroid disease known as "cirrhosis."

In another part of the work, speaking of chronic pneumonia, Laennec regarded the condition as decidedly rare, but admitted that, though rare, it did sometimes follow the acute variety. He mentioned two forms of chronic pneumonia, a red and a grey induration. Elsewhere, in treating of tubercle he described the induration so often observed around tubercles and tubercular cavities, under two forms, the grey tuberculous infiltration, which was tough, smooth, of the consistency of cartilage, and eventually broke down into caseous tubercle; and the jelly-like, tuberculous infiltration "like unto an unusually viscid œdema," but which also broke down into caseous tubercle. In opposition to the views of Andral and Chomel, who believed this zone of grey tuberculous infiltration to be nothing more than chronic pneumonia, Laennec held that though often mistaken for such, it was yet in reality tuberculous.

It will be seen from his cases, that among the symptoms presented by individuals affected with bronchial dilatation, Laennec gives long-continued cough and expectoration with hæmoptysis, and later, diarrhœa, hectic, wasting, and sweating, with occasional œdema. We gather then, from Laennec, some well-described cases of fibroid induration of the lungs occurring often with dilatation of the bronchi, with thickened pleura and contracted lung, and with the occasional presence of cavities. We think we may assume with later writers, that the condition

described by him as occurring round tuberculous cavities and masses, was, in reality, of a fibroid nature, which, eventually becoming infected, did at last become tubercular.

Andral.—On turning to the equally important work of Andral, his *Clinique Médicale*, published in 1823, there is found much bearing, directly or indirectly, on the subject. As one might suppose, it would have been almost unaccountable if, in such an enormous mass of clinical material, there was not some mention of a condition resembling what we understand as fibroid disease. Andral, in relating a series of cases, illustrating the clinical course of bronchitis, mentions a case which was complicated with melanosis. Briefly, the main points of the case were as follows: A cook, aged 65 years, had suffered from dyspnœa, cough and expectoration, for ten or twelve years. The expectoration used to come up in gushes; but he never had fever, emaciation, or night sweats. At the autopsy, in addition to the lungs being œdematous, and presenting the ordinary signs of bronchitis, there were areas of hard, non-crepitant lung-tissue, which cut with difficulty and were the seat of a black deposit. This condition, Andral called "infiltrated melanosis." There were no bronchial dilatations in this case. We must remember that melanosis goes hand in hand with the chronicity of the case. The greater the chronicity, the darker the colour.

Elsewhere, in speaking of chronic inflammation of the lung, Andral described several states of induration around tubercles, where it was in colour, red, grey, or, in extreme cases, black. He considered it easy of proof that the red, grey, and black varieties were the result of chronic pneumonia, and maintained that Bayle's phthisis with melanosis must be looked upon as a chronic inflammation, in or around which a deposition of black colouring-matter had taken place. He further held that Bayle's granular form of phthisis with melanosis, was nothing more than indurated lobules, which had become the seat of black pigment. Andral says that these states of the lung can be met with at all periods of life, and that it is uncommon to find no tubercles in a lung affected with melanotic induration. It will be noted that Andral's views are in opposition to those of Laennec. Laennec held all the chronic processes going on around tubercle to be essentially tubercular. Andral, on the contrary, believed them to be the result of chronic pneumonia. He also advances a step beyond the conception of

Bayle when he ascribes the cause of melanotic induration to chronic pneumonia.

In Andral's remarks upon the pathology of pneumonia, we meet with a still more advanced development of his views. In chronic pneumonia, he distinguished two advanced changes, a red hardening, and a grey hardening or induration. He stated that in healthy lungs, the interlobular tissue was not visible, but that in a lung affected with chronic inflammation, it is seen as thick, white lines of cartilaginous appearance. The rarity of this condition, as a sequence to the acute lesion, he admitted, but had seen it, more frequently than Chomel, affecting sometimes a whole lobe, sometimes a lobule. The common way for chronic pneumonia to arise, according to Andral, was idiopathically. The original site of the change, he thought, was probably in the air-sacs, and from thence it spread to the interlobular tissue.

These opinions of Andral are more definite than those of either Laennec or Bayle. He states his belief as to the starting-place of chronic pneumonia, and as to the mode of origin. Regarded in the light of a contribution to the history of fibroid disease in general, it is perhaps somewhat incomplete and doubtfully accurate, but he at least deals with one form of the process, which is held to be a chronic pneumonia.

When this chronic form does occur with tubercles it may have preceded them, or may have been a consequence of their irritation.

Chomel, writing in the *Dictionnaire de Médicine* in 1827, gave an account of chronic pneumonia. We have already quoted his opinion as to the real nature of the cases published by Broussais. To mark the rarity of this affection, at any rate as a sequel to the acute form, Chomel says that out of 125 cases of acute pneumonia, only one passed into the chronic state. He mentioned the indurations found around tubercles, cavities and gangrenous patches, and further on described chronic pneumonia itself, as a condition in which the lung is blackish-grey, dense, hard, and firm, resisting traction and pressure, and non-crepitant; sometimes slightly granular, at other times smooth, with white bands marking the increase of interlobular tissue.

Out of the eight cases upon which his observations were based, in five the lower lobe was affected, in two, the middle and superior lobes, and in one case the whole lung was involved. The disease

apparently was found as often on one side as the other. The pleuræ were adherent and thickened, more especially in the upper part of the lung. As regards the age, all his cases were over 20 years, and all but one under 50. Chomel was unable to state anything with precision as to the cause, except that when occurring as a sequel to the acute form, it was due to some debilitated state which checked its proper resolution, and he further thought that resolution, even of the chronic form, might sometimes take place.

Chomel confines himself chiefly to the appearances after death, and beyond accurately stating the part of the lungs most commonly affected, and clearly giving the ages of the patients, he adds but little to the accounts of those writers coming before him. It will be observed that, although he calls this state chronic pneumonia, yet, in common with so many others, Chomel almost doubts, though he admits the possibility of, its arising after acute pneumonia.

Stokes.—After Chomel, little of importance was done in fibroid disease until 1837, when Stokes published his work on Diseases of the Chest. On page 137 of that work, he relates a case of bronchiectasis, in which a man, aged 40, had been subject to cough ever since he was a boy. On admission to hospital, he had bronchitis, with attacks of orthopnœa coming on in paroxysms, the cough being attended with copious expectoration. On inspection, the right side was noticed to be very convex, and on percussion, hyper-resonant. There were sibilant *râles* to be heard over this side. The left side in its lower two-thirds was dull on percussion, and here there was an unusual degree of vocal resonance. The heart's impulse extended over a wider area than was natural, being seen in the epigastrium and to the right of the sternum. At the autopsy, the whole of the right lung was found to be in a state of emphysema, the lung being bulky and extending over to the left side. The anterior edges of the left lung were similarly emphysematous, though only to a slight degree. The left lung was much diminished in size. Bronchial dilatations were found throughout the whole of the lung, and the intervening pulmonary tissue was hard, and contained small abscesses.

This case can surely be viewed in no other light than that of a case of fibroid induration of the left lung, with co-existing bronchial dilatations, and emphysema. But Stokes put a very different interpretation on the clinical facts before us. He seemed to consider

the emphysema of the right lung to be more of a primary, than a secondary, condition. The trifling emphysema of the left lung he adduced in favour of this view, maintaining that also to have been the remains of a widely spread emphysema of the left lung. All this condition he ascribed to the result of bronchial dilatation. He did not attribute the whole process to the effects of fibroid disease of the left lung, and were this case to be described now, it could hardly pass under any other denomination.

Corrigan.—A year after this account was written, another Dublin physician, Sir Dominic Corrigan, gave a very minute and precise account of a form of fibroid disease, in the *Dublin Medical Journal* of 1838. Corrigan described a disease which resembled phthisis, but which he strenuously maintained was *not* phthisis. He sought an analogy in the changes taking place in the liver, to which the name "cirrhosis" is applied, and believing the same agencies to be at work in the lung as in the liver, he proposed to style this disease "cirrhosis of the lung." Corrigan taught that the main feature of cirrhosis was a formation of fibroid tissue in the interlobular septa of the lung, and around the bronchi, which tissue, in growing, gradually replaced the pulmonary substance, and finally contracting, drew apart the walls, and so dilated the bronchial tubes. Thus the dilatation of the bronchi was merely secondary to the fibroid process taking place in the lung. The lung thus became very much diminished in size, and eventually consisted only of fibrous tissue and dilated tubes. Corrigan's idea of cirrhosis was, that it was primarily an interstitial process, and had nothing to do with the alveoli of the lungs. According to him, there are three structures in the lung which favour the contracting process: 1, "cellular tissue"; 2, "the fibrous envelope of the lung"; 3, "the elastic tissue of the bronchi."

He gave three complete cases illustrating this condition; two of them came under his own observation, whilst the other was that which has already been quoted above, when speaking of Laennec's cases. Besides these, he mentioned instances of several other persons who, at the time of his paper, were still living.

His first case was that of a child. Three months before, it had contracted influenza, and at the autopsy, the left lung was quite sound; there were no tubercles to be seen anywhere; but the right lung, when cut into, presented the appearance of being intersected with white bands of fibrous tissue, and the bronchi

were dilated. In his second case, the disease had lasted as long as thirty years. The right lung was affected, and the heart was displaced to that side. In this case also there were no tubercles. The third case which he relates was Laennec's third, quoted above.

Among the physical signs, he noted in these cases, gurgling and crepitation, and pointed out that these signs have led people to mistake cirrhosis for advanced phthisis. As further evidence against this disease being phthisis, he instanced the absence of constitutional *wasting*, and insisted on the quiet pulse and the long duration of the case as being altogether opposed to tuberculosis. The publication of these cases and the hypotheses based thereon, excited an almost virulent opposition in many quarters, and among those most at variance with these views we have to mention the names of Stokes and Graves.

But nevertheless, the conclusions of Corrigan soon received wide and general acceptance, and the name he gave the disease has been adopted by all subsequent writers. It will at once be seen, however, that Corrigan's cirrhosis has little to do with other forms of fibroid transformation. In reality, he was describing only one part of the disease, that which begins in the interlobular tissue, not in the alveoli themselves, as in chronic pneumonia. Some authors, among whom may be mentioned Sutton, look upon cirrhosis as a very advanced stage of fibroid degeneration; while others regard it as the result of tubercle which has undergone obsolescence. However that may be, Corrigan's description has formed the text for all writers who have dealt with this part of the subject; and we are indebted to him for this remarkably clear statement, and for the recognition of the signs and symptoms which enable one to diagnose cirrhosis of the lung. It may be noted that in this account we first hear of displacement of the heart and other organs as a result of the contracting process going on in the lung.

Addison.—Between the years 1840–45, several writers of great eminence directed their attention to this fibroid condition of the lung. Foremost among these was Addison. In speaking of lung indurations in general, he described his three well-known varieties: the "uniform albuminous induration"; the "granular induration"; and the "grey induration"; all three of which he considered to be the consequences of acute pneumonia, either slow to resolve, or coming on in repeated attacks. But he doubted altogether the origin of induration in chronic pneumonia. These three varieties

differ in their appearance, colour, and power to become organised—the uniform albuminous kind being most highly organised, the granular form showing little or no tendency to become so, and the grey variety being capable of only partial organisation. Addison maintained that the uniform albuminous induration never was, and never did become, tubercular. He held that the other two varieties, although sometimes becoming tubercular, nevertheless did often undoubtedly occur uncomplicated with tubercle. According to him, this affection, when occurring without tubercle, was distinguished from ordinary phthisical lesions in three ways: 1, by evidence of past inflammation, derived both from the history of the patient, and the puckered and adherent condition of the pleuræ and lung; 2, by being found most commonly in the lower lobes, whilst tubercle generally occurred in the upper; and 3, by the total absence of tubercle in other parts of the body. Later on, he maintained that these indurations might soften, become caseous, or break down into cavities; and quoted cases where caseous deposits were associated with induration, but in which no trace of tubercle existed. Addison also gave accounts of cases of tubercle on the way towards organisation, surrounded more or less by the darker forms of this induration. Under the designation of "pneumonic phthisis," he narrated several cases of typical fibroid disease, in which the autopsies showed indurations, dilated bronchi, with usually no cavities, and never any tubercles; whilst under the title of "*tuberculo-pneumonic phthisis*," he clearly laid down the coincidence of reparative indurative changes, surrounding, and associated with tubercle, which changes, resulted from pneumonic inflammation; but he drew no distinction between cases originally, or only secondarily, tubercular.

Whilst discussing the pathology of phthisis, Addison again reverted to the indurations left behind by a pneumonia; and stated his firm conviction that whether they occurred in tuberculous lungs or not, they had the same origin—viz., in pulmonic inflammation; and he considered these indurations might take on the course of scar tissue; in other words, they might slough, soften, break down, form cavities, become calcareous, or remain more or less fully organised and quiescent. Lastly, in speaking of physical signs, he believed that chronic induration with dilated tubes could not be distinguished, by signs alone, from tubercular phthisis.

All through Addison's works, one is constantly meeting with cases which were diagnosed and looked upon ordinarily as cases of phthisis; yet which, owing to the tenacity wherewith this writer followed up his observations to the end, led him to the firm conviction that cases did occur which, though presenting many, if not all, the physical signs of tubercular phthisis, yet differed from that disease, *before* death, in their chronicity, in the absence of wasting and sweating, and *after* death, in the appearances met with at the *post-mortem* examination. The symptoms which Addison found mostly associated with these conditions were those of chronic bronchitis, such as cough and expectoration. Sometimes, there was hæmoptysis, but rarely was there so much constitutional disturbance, in the way of hectic and emaciation, as in the ordinary forms of phthisis. The physical signs most common were those of an unresolved pneumonia—viz., widespread infiltration, consolidation, and excavation. He was struck, in one or two cases, with the very wide extent of the physical signs, and the paucity of constitutional symptoms; and this is exactly in accordance with modern ideas.

We must not leave Addison, without searching his writings for some opinion as to why these changes and conditions should occur: why one case of pneumonia should completely resolve, and another remain more or less unresolved, and tend to undergo either organisation or degeneration. Concerning this point, he says that pneumonias have a tendency to remain unresolved in persons of cachectic and strumous constitutions, or who are in a state of debility or low vitality, and that afterwards the unabsorbed exudation becomes more or less organised. But he offers no explanation why in one case there should be a large, and in another a small, amount of the indurative and reparative processes. Indeed, from his writings, one would be led to take it as his opinion, (1) that pulmonary indurations were almost always the result of pneumonia or of pulmonic inflammation of some kind, at first free from tubercle; and (2) that when found with tubercle, the latter had become engrafted secondarily.

Grisolle, writing in 1841, in his well-known work on Pneumonia, regarded the condition of pulmonary induration as due to chronic pneumonia, and divided it into two classes: 1, The simple form, which occurs independently of any other condition; and 2, the tubercular form, which occurs around crude tubercles and

cavities.* The simple variety, according to Grisolle, may arise as the result of an unresolved pneumonia, or independently. In the first stage, Grisolle described simply a firmer condition of red hepatisation, in which the lung is infiltrated with a fibrinous exudation, and this he held to be only a modification of the acute stage. Later, when the condition is well advanced, he described a red and grey induration much as does Laennec, but did not use these terms. The lung-tissue is increased in weight and diminished in size, difficult to break down, at times granular, and the white lines of interlobular tissue are much increased. There can be no doubt as to the meaning of the author, from his description of the indurated and fibroid condition of the lung, whatever the opinion may be as to the origin of the affection.

Let us now see what Grisolle says concerning the changes in the minute anatomy. With regard to this point, he quotes largely from Charcot, who says that, "there is an amorphous granular material effused into the vesicles and alveolar walls." Entangled fibro-plastic elements (sometimes nucleated) and granular corpuscles are also found. The lining epithelium of the "vesicles" becomes granular, sometimes separates from the walls, or may altogether disappear.

In a later stage, the alveolar walls may be seen very much thickened, sometimes to three times their original size. The fusiform fibro-plastic cells are now replaced by more or less well-formed connective tissue.† The vesicles themselves then tend to become effaced, and their contents absorbed. The capillary vessels in a great measure are obliterated, a considerable amount of pigment is deposited, and the pulmonary parenchyma appears almost entirely composed of connective tissue.

Grisolle then goes on to consider the various retrograde changes which may take place in this indurated tissue. He mentions fatty degeneration, softening and suppuration, and quotes a case of chronic abscess of the lung, surrounded by induration. Further, he mentions gangrene as an occasional result of this process, and enlarges upon the great difficulty there often is in

* But it must be remembered that this second variety can hardly be regarded as a parallel to the first variety.

† But while this is true for the chronic forms of pneumonia, yet in acute pneumonia there are no evidences of textural development.

deciding which is the initial change, the abscess and gangrene, or the induration.

With regard to the extent to which chronic pneumonia may occur, Grisolle had seen it invade part or whole of a lobe, or a whole lung, or even affect both lungs to a greater or less degree.

Grisolle did not think the induration favoured bronchial dilatation, although he allowed that the latter affection occurred with chronic pneumonia; he criticised the opinion of Durand-Fardel adversely, and held that the dilatations probably preceded the induration. Again, although Grisolle admitted the pleuræ to be often affected, yet he would not allow them to be more so in the chronic than in the acute variety. Lastly, he regarded cirrhosis as essentially the same change, and distinctly stated that in the condition he was describing, there was absolutely no tubercle in the diseased or in the sound lung.

Grisolle's is mostly a pathological description; and beyond stating that when the disease is well established, the chest wall falls in, he says little or nothing about the cause, physical signs, or symptoms. So, too, we gain but little with regard to the antecedents of this condition, except his statement that syphilis may give rise to similar changes, and that this lesion may be caused by working in carbon, or other forms of irritating dust.

The indurations around tubercles he considers to be due to chronic pneumonia, often, of course, becoming infected and breaking down with the tubercle; but he does not regard them as primarily tubercular, as does Laennec.

Hasse.—At about the same period, Hasse published his "Pathological Anatomy," which was afterwards translated in the publications of the Sydenham Society. In speaking of chronic pneumonia, Hasse says: "There is a state after acute pneumonia, in which the air-cells fail to recover themselves, the effused substances becoming amalmagated with the parietes of the alveoli. This obliteration of the cells is connected with vascular development in the effused product. The lung then gradually shrivels; the walls of the thorax sink in, and dilatation of the bronchial tubes results." Chronic pneumonia he held to be rare, and scarcely ever seen as a sequel to acute pneumonia. Hasse assented to Andral's teaching that red and grey induration were nothing more than chronic pneumonia, and remarked that in the few instances

he had seen, the indurated portions of the lung were at the base and tubercles at the apex. Hasse then goes on to quote Hope and Andral, who said that "chronic pneumonia is a slowly developed hypertrophy of the septa of the lobules, attended, during the period of augmented vascular activity, with a deposition of albuminous matter in the interstices of the pulmonary substance. The septa are thus thickened and eventually become cartilaginous."

There is, perhaps, not much that is new in this account, but it admits both an interstitial and an intra-alveolar origin, which is more than had been granted by previous authors.

Rokitansky.—Still more decided views upon the subject are taken by Rokitansky, in his great work, published in 1849. He there says that chronic pneumonia is very rare after the acute disease, and that, even when acute pneumonia runs a chronic course, the process is essentially different from that taking place in interstitial pneumonia. Speaking of interstitial pneumonia, he stated that, although the air-sacs were frequently implicated, yet the interstitial tissue proper was the chief seat of the process, it being infiltrated with an albuminous substance which became organised, and in time formed a dense fibro-cellular substance. This condition he thought most common in the upper lobes.

In the two preceding accounts, we hear for the first time (with the exception of Corrigan's paper) of the process being in the main an interstitial one, although the air-cells may and do become secondarily affected.

Handfield-Jones.—In 1854, several books appeared, each dealing directly, or indirectly, with fibroid degeneration taking place in different tissues of the body. Reviewing these books in an elaborate and thoughtful paper, entitled "Fibroid and Allied Degeneration," in the *British and Foreign Medico-Chirurgical Review*, Dr. Handfield-Jones sought to link together the fibroid processes going on in the body, and to assign them all to a general constitutional cause. Where one organ alone was attacked with fibroid change, he held that it was but a local manifestation of a general disease. The fibroid disease of the liver, termed cirrhosis; that of the kidney, known as interstitial nephritis; the fibroid condition met with in the pericardium, heart, and valves; the various forms of degeneration taking place in the brain, and the chronic changes occurring in the lungs and pleuræ, were, according

to Handfield-Jones, all due to a fibroid diathesis, falling with greater or less intensity upon some organ of the body, whose nutrition was sufficiently impaired for the onset of the process.

He cited many cases in which there was evidence of two or more organs of the body being affected at the same time. He believed this general constitutional disease was due to an unnatural state of the blood, giving rise to unhealthy exudations, and suggested that the growth of fibroid tissue, though started perhaps, by inflammation, yet might continue irrespective of the latter. He believed that the amount of fibrin exercised a profound influence upon the future of the fibroid change.

Turning to lung fibrosis, he admitted three separate conditions: 1, ordinary hepatising pneumonia; 2, chronic pneumonia, in which the vesicles were filled and also the interstitial tissue affected; and 3, cirrhosis, in which the interstitial tissue was affected primarily, the vesicles being sometimes secondarily involved.

The author of this valuable paper takes a broad and comprehensive view of the question of fibroid disease. In this article, we meet with the first attempt to place so many diverse conditions in one group under the name of "fibroid degeneration." Here we first hear of the possibility of a constitutional origin of fibroid disease in general. One may perhaps, take exception to the word "degeneration"; but, after all, the whole process of fibroid formation anywhere is a substitution of a tissue of a lower form for that of a higher one, and, viewed in that light, is assuredly a degeneration.

Bennett.—All the authors hitherto mentioned have assumed that the fibroid process, whether described under the name of fibroid induration, or chronic pneumonia, was essentially a non-tubercular process. In 1856, however, Dr. Hughes Bennett published his work on Pulmonary Phthisis, and in it recorded a typical example of cirrhosis of the lung. The specimen is now in the Edinburgh Museum. There were a few questionable tubercles at the apex. The lung was the size of the two fists, and consisted mainly of dilated tubes and fibroid tissue. Yet, notwithstanding the facts of the case and the clinical course of the disease before him, Dr. Bennett had no hesitation in saying that the case was simply one in which the tubercles had undergone obsolescence—in fact a case of cured tuberculosis.

Sutton's paper on Fibroid Degeneration appeared in the

Transactions of the Medico-Chirurgical Society in 1865, and, since in it the minute morbid anatomy and general pathology of the process were first discussed fully in England, it will be well to dwell at some length upon the subject-matter of the paper.

Sutton understood by "fibroid degeneration of the lung," a condition in which it is firm, tough, heavy, and sinks in water. The cut surface presents a smooth appearance. Not unfrequently, as the fibroid tissue approaches the healthy part of the lung, a granular condition is noticed, each granule being situated amidst augmented interlobular tissue. Sutton quoted Addison as saying that these granules were not due to tubercle, but to the new fibroid material puckering up the air-cells.

Sutton held that the interlobular connective tissue was much increased. The pleura, as a rule, was thickened, and in the augmented connective tissue there was a deposition of pigment, giving rise to an iron-grey coloration of the lung. He found both lungs to be commonly affected; if one lung alone was attacked, it was generally the left. The process appeared to him to begin most frequently in the upper lobes. He did not find the bronchial tubes markedly enlarged. Finally, Sutton believed the lung change to be usually associated with similar changes in other organs; such as the liver, kidney, heart, spleen, &c.

Cavities were met with in these cases which were deemed to be either the results of softening, or dilatation of the bronchial tubes.

The microscopical appearances are described at length, and with the exception of Charcot and Robin, this is the first account of this branch of the subject.

In the diseased portions, the air-cells could be seen to be obscured and obliterated by a newly formed fibre-like tissue, which was made up of spindle-shaped fibres, with the elastic tissue of the air-cells crossing the new tissue in various directions. A granular material obscuring the arrangement of the fibres above described was frequently observed. Sutton held this to denote the fibroid material in a state of degeneration. He thus summarised the results of microscropical examination:

1. There had been a formation of new tissue elements, (fibroid tissue).
2. This tissue had invaded and destroyed the lung substance.
3. These elements were most highly developed in tissue around

the bronchial tubes, lobules and thickened pleura, and had apparently begun there, and thence extended in all directions until the cells themselves were filled up and obliterated.

Sutton thought that the cheesy masses referred to above, were caused by a degenerative metamorphosis of the fibroid tissue, due to imperfect nutrition. The same cause was assigned to the formation of cavities at the apices of the lungs, and elsewhere. From the cases quoted in support of his views, it appeared that the sufferers were well-built persons. There was generally a history of intemperance, and winter cough had supervened for years. In these cases there was little wasting; the appetite remained good; there was an absence of intestinal disease, and the immediate cause of death seemed to be capillary bronchitis, pleurisy, or bronchopneumonia. Finally, Sutton adduced six reasons for not regarding this condition as one of "scrofula":

1. Microscopically, there were well-formed cells.
2. Cheesy matter is not proof positive of scrofula.
3. Patients were well-built.
4. There were no tubercles, except granulations.
5. No tubercular disease of the intestines.
6. Different tissue was affected to that involved in scrofula.

Recalling how often the lung change was associated with similar changes in the liver, heart, and kidney, Sutton inclined strongly to the belief in a fibroid diathesis, and thought that in order to convert this diathesis into actual disease, an exciting cause, generally to be found in excessive spirit-drinking, was necessary. The disease was prone to occur in the middle and advanced periods of life.

The microscopic account given in this article is in the main exact, and substantially correct. On looking through the cases, however, supplied by Sutton, one can hardly come to any other conclusion than that many of them were cases of chronic tubercular phthisis with a large amount of fibroid tissue superadded; and again, his arguments against scrofula would hardly be accepted now. It seems reasonable to suppose that Sutton was describing in reality the fibroid element of chronic tuberculosis. Yet the fibroid state he described, though taken from tubercular cases, is identical with the process going on in cases without this complication.

Clark.—In 1868, Sir Andrew Clark gave a detailed description of a case of fibroid disease of the lung, and whilst admitting the

occurrence of indurations with tubercular disease, and pointing out that originally non-tubercular trouble might become tubercular, contended strongly that there should be made a broad distinction between this well-marked form of phthisis (to which he gave the name of Fibroid Phthisis), and that ordinarily so called, both clinically and pathologically.

In his definition of Fibroid Phthisis he included all those cases, local or constitutional in origin, which are characterised by the existence of a contracted and indurated lung, traversed usually by more or less dilated bronchi; exhibiting an excess of fibroid tissue, and a tough fibrogenous substance, with cheesy deposits or consolidations, and usually small cavities, found most commonly towards the middle or base of the lung.

In speaking of the causes of this affection, he divided them into constitutional or general, and local. Among the former, he noted the general constitutional tendency of fibroid degeneration, and added other causes which might accelerate, aggravate, or even initiate that tendency; such as abuse of alcohol, syphilis, rheumatism or gout, and in these cases there was evidence of these diseases, or of fibroid degeneration in other organs. Amongst the local causes he enumerated exposure to irritating dusts, tubercle, bronchitis, pleurisy, pericarditis, or unresolved pneumonia.

The case reported with the paper was that of a woman, aged 28, who, having suffered from ascites three years previously, had for four or five months been subject to symptoms of cough with expectoration, hæmoptysis, vomiting, diarrhœa, and wasting. The cough often culminated in vomiting, and was attended by a large quantity of expectoration, often fœtid, which was found to contain elastic areolæ. The physical signs were those of consolidation and excavation at the left base with contraction of the chest, the heart being displaced upwards and outwards, and uncovered. A systolic bruit was heard over the pulmonary artery. The right lung was enlarged and emphysematous, only a few crepitations were to be heard at the base, and these were thought to be conducted. At the autopsy the right lung was found to be enlarged and emphysematous, but otherwise healthy. The heart was displaced, the valves were not diseased, whilst the pericardium was thickened and adherent to the lung. The left lung was contracted, universally adherent, and the pleura much thickened, presenting both old and recent adhesions. Fibroid septa were found traversing

the lung in all directions, but chiefly following the course of the bronchi and blood-vessels, and running from the thickened pleura. The intervening lung was hard, resistant, and in some positions presented caseous masses and some small cavities. Long irregular cavities were also found running from the root towards the periphery, lined with cheesy matter, and one of them resembled a dilated bronchus. Some of the bronchi were dilated; but they were not generally so; their mucous membrane being thick, vascular, ulcerated, and villous.

The microscopical appearances of portions of the lung were as follows: Some of the air-cells were emphysematous and atrophied, others were filled with an amorphous material, showing but little fibrillation, and containing for the most part no cells, though occasionally a few were seen chiefly against the walls of the alveoli, and pus-like or epithelial in character. The fibroid material presented the characteristics of more or less well-formed connective tissue, and was noticed to be most abundant around the vessels and tubes, and springing from the pleura on the surface and between the lobes. The walls of the blood-vessels were observed to be distinctly thickened, (the thickening appearing mostly in the external coat), and some had become impervious. Sir Andrew Clark considered that the changes were due to a fibroid degeneration of normal textures, and that there was no independent production of fibroid tissue in the alveoli themselves, they being invaded from without by the ever-increasing interlobular tissue. Speaking generally, he believed that when the disease was non-tubercular, nearly always one lung only was affected, and the base more commonly so than the apex.

In speaking of the physical signs, he laid great stress upon the almost universal occurrence of contraction of the chest, and displacement of the heart towards the affected side. And with regard to the symptoms, he insisted on the importance of recognising the absence of marked constitutional disturbance and hectic; the slow progress of the cases with the occasional supervention towards the close of œdema, albuminuria, and diarrhœa; the last of which he considered to be due, in some cases, to a non-tubercular ulceration of the intestine.

Wilson Fox. — This observer wrote an article on chronic pneumonia in Reynolds' *System of Medicine*. The views in that article were based on an analysis of 39 cases, of which 22 were

males and 16 females. Two-thirds of these died before the age of 40. Fox believed that in reality the greater number could be classed as either tubercular, or at all events as coming under the head of "cured tubercle." He supposed that the condition might arise from four separate states—viz., pneumonia or broncho-pneumonia, pleurisy, interstitial chronic inflammation, and fibroid changes in the walls of the alveoli. Anatomically, two conditions were described:

1. A *red* induration, in which there is granular thickening of the alveoli.
2. A *black* induration, where the granular appearance has gone, and the lung is tough and friable.

The bronchi were found dilated in 31 out of the 39 cases, and it was not of rare occurrence to find secondary inflammation of the indurated parts.

With regard to the position, Fox found the disease limited to one side in 31 out of 39 cases. The right lung he found affected in 10 cases, the left in 14, the base in 8, the apex in 3, and a double affection of the apex in 3 cases. Bronchiectasis, he thought, might arise either simultaneously with the broncho-pneumonia, precede this, or come after it.

When speaking of the pathology of the affection, he maintained that there was no analogy between it and cirrhosis, holding, as he did, that the chief change in the lung was in the walls of the alveoli. Wilson Fox would not admit a fibroid diathesis, in spite of the fact that he found evidence of fibroid change in the heart and liver in six, and granular changes in the kidneys in twelve cases.

Wilson Fox's views on tubercle are well known, and in describing this disease under the name of chronic pneumonia, he was endeavouring to show that there was little difference between this state and chronic tuberculosis. His descriptions of the anatomy and pathology do not agree with those of Clark, Sutton, and others.

Bastian.—Dr. Charlton Bastian contributed to the Pathological Society's *Transactions*, and to Reynolds' *System of Medicine*, articles entitled "Cirrhosis of the Lungs," in which he gave a most careful analysis of 30 cases of this affection, collected from various sources, some previously published under somewhat similar titles, and some recorded by himself.

Using Corrigan's term "cirrhosis," he quoted that writer's opinion

as to the formation of bronchiectasis, and proceeded to distinguish cirrhosis from tubercular affections, and also from bronchiectasis. In order to mark the difference between cirrhosis and this latter affection, he instanced the different ages at which they occur. Twenty-six out of 43 of the cases of bronchiectasis occurred after the age of 60 years, whilst almost two-thirds of the cases of cirrhosis were found between the ages of 15 and 40 years. Further, Dr. Bastian considered it almost always the rule to find one lung only affected in cirrhosis, while this rule by no means held good in bronchiectasis. Lastly, he stated that hæmoptysis was more frequent in cirrhosis than in bronchiectasis, and that whereas the former affection is more common in males, the latter falls with greater intensity upon females.

In treating of the minute anatomy, authors are quoted as writing on the same disease under different titles, as we have seen before in our review of their researches. Attention is drawn to the fact that they all agree as to the occurrence of a fibroid metamorphosis, which may be chronic from the first, or occur as a sequel to acute pneumonia. Dr. Bastian was inclined to agree with these authors in the main, but objected to the terms used. Thus, whilst he agreed that the process described by Rokitansky and others, under the title of interstitial pneumonia, would give rise to this condition, he objected to the word pneumonia being employed, since in it there is no pneumonic exudation. Similarly, although he admitted the process described under that name, he objected to the title of chronic pneumonia, in that the process—

1. Pathologically, is quite distinct from pneumonia.
2. Though sometimes a sequel to it, is yet quite different.
3. May arise independently of any pneumonia, and

Lastly, that there is no inflammation, but simply the formation of a new overgrowth.

It does not appear that Dr. Bastian does away altogether with inflammation as part of the process, but he points out most clearly that this cirrhosing process differs essentially from ordinary inflammation, in that there is no destruction of the granulation or lymphatic tissue first formed, but that it is organised, and persists as a developing, fibroid growth. Likewise he distinguishes it from a degeneration, because a new tissue is formed, not an old one degenerated. This is true in a measure, but, as has been before remarked, this new tissue is formed at the expense of the more

highly developed pulmonary tissue, and the process, in that sense, is a degeneration.

In speaking of the various positions in which this process may occur, Dr. Bastian enumerated the connective tissue of the walls of the bronchi, the same tissue spread around blood-vessels, the existing interlobular tissue, or the inner surface of the pleura; but he made no mention of any affection starting from the alveoli themselves.

Bastian described the naked-eye appearances of the lungs, such as, from the descriptions of former writers, we have come to look upon as quite typical. But he explained more clearly than we have formerly noted, the origin of the ulcerated caverns. He says that without tubercle they occur in one-fourth of the cases, and may originate in three ways: either by fatty degeneration of the new tissue, or by its becoming gangrenous, or by spreading ulceration of the tubes.

With regard to the antecedents of these cirrhosed lungs, Bastian admits local and general causes, and although he allows this condition may occur in connection with a general fibroid diathesis, he does not consider it to be simply a local manifestation of a general disease; for, as he says, it can, and does, occur without any general fibroid tendency whatever.

In the analysis of the very valuable cases he places on record, we may note some most interesting points.

First among these stands out the frequency with which hæmoptysis occurs, viz., in 17 out of 30 cases; in only two of these 17 could tubercle account for it.

(2) Equally important is it to bear in mind the points learnt with regard to contraction of the chest—viz., that it is always present after eighteen months' duration of the disease, that it may occur even before, in people with very flexible chests, and that it is always in inverse proportion to the amount of displacement of organs and bronchiectasis. That is to say, that when the fibroid lung contracts, it pulls the chest walls in, or the walls of the bronchial tubes out, or the surrounding organs towards it, and in different cases these various conditions will be found combined in varying proportions.

(3) The large number of cases in which the heart is affected is also worthy of attention, marked hypertrophy, especially of the right side, being found; and in three cases complicated with tricuspid regurgitation.

Lastly, those instances in which the pulmonary artery is noted as being narrowed, atheromatous, plugged, or entirely obliterated, are the very cases which have advanced to such a degree of contraction, that the lung has become a mere appendage, impermeable to air, useless and unused.

Iuergensen.—An extremely instructive article written by Iuergensen, appears in Ziemssen's *Cyclopædia of Medicine*, under the title of "Interstitial Pneumonia." Iuergensen perceived the difficulty of dogmatically defining the process, and truly says that the greater part of this difficulty lies in the fact that interstitial pneumonia never occurs as an independent affection, though, as an accompaniment of various common bronchial troubles its occurrence is fairly frequent.

He mentions the two schools of opinion as to the formation of bronchiectasis, the one led by Laennec and the other by Corrigan—the former regarding it as the primary, the latter as the secondary, affection. Further on, we gain some information with regard to the causation of lung induration. Iuergensen is definitely of opinion that chronic bronchial catarrh is capable of setting up interstitial changes, either through the decomposition, and resulting irritating character of the secretion, or even by its simple chronicity. In connection with this point, he instances the common occurrence of this affection in the lungs of those exposed to the inhalation of irritating forms of dust. With regard to the formation of the interstitial changes, he adds the still more interesting explanation, that it is due to impaired function, and lymphatic flow, in a lung where the interstitial tissue is blocked by more or less irritating foreign bodies. This is a new thought as to the causation and perpetuation of this process, for, we can understand that a lung or a portion of a lung, bound by adhesions, or in any way prevented from undergoing its normal movements and expansion, will be placed at a disadvantage as to its recovery, if it should become inflamed or catarrhal, and thus the interstitial changes may be originated. Moreover, when once started, the resulting impairment of lymphatic flow will still further tend to perpetuate that change. Iuergensen gives the age at which this affection most commonly occurs as between 40 and 60; but says that it nearly always *originates* in childhood, from which we gather that it often takes some years to arrive at such a pitch as to cause symptoms. Catarrhal pneumonia in connection with

measles and pertussis occurring in childhood, he regards as by far the commonest antecedent. When the disease begins later in life, pleurisy and bronchitis are, according to him, the chief causes, and he admits croupous pneumonia and syphilis as occasional antecedents. According to Iuergensen, tubercle may be deposited secondarily, but the induration may exist for a long time without its supervention. The most usual position for the induration is at the base, except when it occurs with primary tubercle, and then it takes the position commonly assumed by that affection, at the apex. The resulting contraction of the lung and falling in of the chest, and the displacement of viscera, are noted. Attention is drawn, as the case progresses, to the usual supervention of cardiac hypertrophy, congestion of the spleen, liver and kidneys, which Iuergensen says are often fatty, but rarely amyloid.

Turning to the clinical aspects of the case, the absence of the ordinary symptoms of phthisis is noted—the good nutrition, the absence of fever, the good, slow, pulse, associated with many symptoms which are met with in that affection, such as the chronic cough and expectoration.

According to this author, death may occur from exhaustion, dropsy, dyspnœa, or from an attack of acute disease in the already narrowed respiratory tissue, or even from a fatal attack of hæmoptysis. In conclusion, Iuergensen states that he never hesitates to exclude ordinary phthisis, if there be great physical or mental activity, or a steady and vigorous action of the heart, and he regards the absence of fever also as an important point in the diagnosis between these two affections.

Powell.—In 1869, Dr. Douglas Powell reported three cases in the *Transactions of the Clinical Society* (vol. vi.), under the title of "Phthisis with contracted Lungs," which are worthy of note.

Case I., aged 40, was admitted suffering from severe hæmoptysis, from which he rapidly died. The history showed that one of his brothers had died of phthisis. Personally, he had suffered from cough for six years, had had pneumonia a year ago, and his first attack of hæmoptysis had occurred six months previously, since which he had had several subsequent attacks, for the last of which he was admitted into hospital, and during which he died. At the autopsy the left lung was found to be contracted, with the pleura thickened, in some places to as much as half an inch. Long irregular cavities, which were thought to be dilated bronchi, were

to be seen running in a direction from the root to the periphery of the lung, and there were also tubercular granulations, and much increase of fibroid tissue throughout this organ. The right lung was enlarged and emphysematous, there was no thickening of the pleura, neither was there any increase of fibroid tissue, but a few tubercles were found at the apex. The heart was found displaced; the intestines were not examined. Microscopically, portions of the left lung showed much increase of fibroid tissue, with the alveoli collapsed, their walls much thickened, and for the most part empty, or at any rate remarkably free from the large granular cells of epithelial pneumonia. In commenting on this case, Dr. Douglas Powell was of the opinion that it was one of old arrested tuberculosis, or one of interstitial pneumonia in which fresh tubercle had supervened, and he distinguished it from "cirrhosis," because in it, there were the physical signs of excavation. But we must remember that, although the signs of excavation are not so common in "cirrhosis" as in other forms of fibroid disease, nevertheless they do sometimes occur, and this distinction would not hold good in any way when all the different forms are taken into consideration, for in them the signs of excavation, due either to cavities or dilated tubes, are the rule and not the exception.

Case II., aged 38, having no history of phthisis in the family, had suffered from an acute illness five years previously, the nature of which was unknown, and had subsequently had an attack of pneumonia, six months before, since which time he had been definitely ailing. The signs here were those of contraction of the left side of the chest, with those of excavation. The heart was displaced, and there was a mitral systolic bruit. There were also signs of slight disease of the right apex. There was no albuminuria. He improved and gained weight, but the disease seemed to increase on the right side.

Case III., aged 57, having no phthisical taint in the family, had had a subacute attack of either pleurisy or pneumonia eight months previously, not severe, and running a chronic course. Here there was no hæmoptysis, and the signs were those of contraction of the left side, with excavation at the level of the second intercostal space in the axilla. The heart was displaced, and there was albuminuria. There was also in this case very doubtful early disease at the right apex.

In all these cases, from the contraction of the chest and from

the displacement of the heart, associated with signs of excavation, there would seem to be but little doubt as to the presence of fibroid disease. In Case I., the *post-mortem* examination showed this to have been complicated with tubercle; and in the other two cases, from the affection of the apex of the opposite lung, one would suspect that they, too, were similarly complicated, though it must be remarked that in the last case the presence of that disease remained doubtful; and in none of them could it be definitely stated which was the primary affection, the tuberculosis or the fibroid disease.

Walshe, in his well-known treatise on Diseases of the Lung, gave an account of chronic pneumonia and cirrhosis. He believed the two conditions to be quite distinct, both clinically and pathologically. Whilst admitting that in cirrhosis the chief seat of change was the interstitial tissue of the lung, and in chronic pneumonia the alveoli, he was yet quite ready to allow that, as a secondary result, the alveoli became implicated in cirrhosis, and the interstitial tissue in chronic pneumonia.

He stated chronic pneumonia to be a rare condition, either as a sequence to the acute form, or as a primary disease; but common enough as a concomitant with chronic tuberculosis. Walshe described two forms: 1, A state in which the lung did not break down; and 2, a state in which, from some cause or other, cavities were formed through softening of the infiltrated lung-tissue.

In his account of cirrhosis he closely followed Corrigan, He believed the formation of cavities to be rare in cirrhosis, and although he met with fibrosis of other organs in association with that of the lung, he would not admit of a diathesis. Walshe saw some cases in which there was good reason for believing the existing cause of cirrhosis to be the abuse of alcohol.

At one time Walshe believed cirrhosis to be antagonistic to tubercle; but subsequent investigation convinced him that the two co-existed much more often than he at first supposed. In the *Medical Times and Gazette* he published a case of cirrhosis, which is, without doubt, the most completely reported case of this disease extant.

Recent Research.—Since 1868, the date of Sir Andrew Clark's paper, very little that is new has been done in this field. Beyond a passing mention in different works dealing with lung disease, and the records of a good many cases to be found in the *Transactions* of

the Pathological Society, the subject, so far as advancement is concerned, has slept.

Indeed, in text-books, the stereotyped descriptions of the disease, as given by Corrigan and others, have been followed closely. It is impossible to quote at any length all the opinions of writers who have touched on the subject in these latter years, nor would it be necessary, since they have drawn their opinions largely from the authors to whom we have already referred at length. The opinions, however, of some of them may with advantage be epitomised.

Fagge, in his text-book described the disease under the name of cirrhosis, and seemed to be of opinion that, to use his own words, "in all but an insignificant minority, the so-called cases of fibroid phthisis are nothing but an advanced stage, or a very chronic form, of a disease that is really tubercular."

Dr. C. I. B. Williams and **Dr. C. T. Williams**, in their work on Consumption, give two modes of origin, one from pleurisy and interstitial pneumonia, another from chronic pneumonia, resulting from the inhalation of particles of dust. They hold to the belief that the disease is essentially tubercular, and maintain that tubercle bacilli can be detected in the sputum. They quote Watson Cheyne and Percy Kidd, as having found in these cases no bacilli in the fibroid tissue, but in large quantities in the cheesy masses, found after death in the lungs. In the second form, that arising from the inhalation of dust, Dr. Williams states that multitudes of bacilli are generally to be found.

Dr. Eustace Smith, in his clinical studies on *Disease in Children*, gives some very typical cases of this disease, and assigns the chief cause to pleurisy and catarrhal pneumonia. He believes both the interlobular tissue and the vesicles to be the seat of the morbid process, and further gives cases in which ulcerative destruction of the fibroid tissue is set up, constituting fibroid phthisis.

Peacock and **Greenhow**, in the *Transactions* of the Pathological Society, give interesting cases of lung disease induced by the inhalation of irritating dusts. Many of these were undoubtedly tubercular, having been set going by the irritating dust; but some, on microscopical examination, appeared to present all the characters of chronic pneumonia caused by the irritation. Greenhow relied on a cool skin, quiet pulse, and a wheezy asthmatic cough, to distinguish this disease from tubercular phthisis. Peacock held that if a man had an hereditary predisposition to tubercle, his occupation,

followed in an atmosphere of dust, would favour the onset of tubercle, whereas if there were no such predisposition, he would be more likely to develop only chronic pneumonia.

Among the various writers in the *Transactions* of the various Societies may be mentioned **Dr. Green, Dr. Barlow, Dr. Bastian, Dr. Fagge, Dr. Coupland, Dr. Wilks,** and **Dr. Percy Kidd,** all of whom give some very excellent examples of the disease in one form or other.

Scattered over general medical literature can be found many notices of cases which have come under the observation of different workers, but the pathology of such cases has already been given.

Lastly, in the *Clinical Journal,* **Dr. Ewart** has published a lecture on the subject of cirrhosis of the lung; and recently a book has appeared by Dr. Auld, entitled *Fibroid Pneumonia,* dealing with this subject.

It will be seen from the foregoing review, how diverse opinions are as to the origin and cause of this affection. We have quoted at length many authors, most of whom differ in their descriptions and deductions. This is not to be wondered at, since so many of them were describing different conditions, all of which we wish to include under the general designation of "Fibroid Diseases of the Lung and Fibroid Phthisis," reserving for the latter title those cases of pure fibroid disease, uncomplicated primarily or secondarily with tubercle, and associated with cavities in some form or another. And since, in these descriptions and in our own collection of cases, there are to be found instances of pure fibroid disease, and cases complicated primarily or secondarily with tubercle, we propose to classify all fibroid disease under three headings:

1. *Pure Fibroid, Fibroid Phthisis,* by which we understand a condition in which there is no tubercle.
2. *Tuberculo-fibroid Disease,* a condition which is primarily tubercular, but has subsequently run a fibroid course.
3. *Fibro-tubercular Disease,* a condition in which primarily fibroid disease has become tubercular.

CHAPTER II.

ON THE TERM "FIBROID PHTHISIS."

THROUGHOUT this book, as well as upon other occasions when writing upon the subject, we have employed the term "Fibroid Phthisis"; and, as it has been widely discussed, inaccurately apprehended, and adversely criticised, we regard it as a duty to our readers to explain the term and to justify its retention.

Assuredly, to invent names and apply them to ill-defined, incoherent, and unstable groups of facts and phenomena, is an offence against scientific method. But, on the other hand, to refuse a distinctive appellation to well-defined, coherent, and permanent groups of facts and phenomena, of which certain characteristics may be universally predicated, is to violate an imperative scientific duty.*

What then, are the criteria of a specific distinction?

If a given group of facts and phenomena is well defined, if it possesses sufficient unity and continuity of history, if its constituents are interrelated and coherent, if it responds in the same general manner to its environments, if throughout its course there can be always predicated of it what cannot be predicated of any other group, then the framing of a specific distinction is not merely justifiable but imperative.

Next in importance to the criteria of distinction come **the conditions of naming.** There are at least three. (1) The name should be simple and intelligible; (2) it should be formed out of the unchanging physical characteristics of the group named; and (3) it should neither convey nor suggest any hypothetical explanation of its nature. Due attention to these conditions would exert the happiest influences upon the course and character of scientific

* "Where a certain apparent difference between things (although perhaps in itself of little moment) answers to we know not what other differences pervading not only their known properties, but properties yet undiscovered, it is not optional but imperative to recognise this difference as the foundation of a specific difference." (Mill's *Logic*, Book I. c. vii. § 4.)

discussion, and greatly promote the progress of clinical knowledge. We fear it is mainly to disregard of them, and to want of precision in the use of language, that we owe our present controversies about this subject, and the personal bitterness which they sometimes beget.

Let us now apply these considerations to the employment of the term **Pulmonary Phthisis.** By this term is meant that assemblage, progression, and relation of signs and symptoms, associated with or dependent upon, the ulcerative or suppurative disintegration of more or less circumscribed, non-malignant consolidation of the lungs. This term neither suggests nor conveys any hypothesis; it is framed out of the physical characteristics of the group of facts requiring to be named; it may be predicated of this group throughout the whole of its history; it is both convenient and intelligible in use and application; it enables discussions to be conducted without misunderstandings; it has the sanction of immemorial usage; and, whatever the direction which knowledge in its progress may take, the term "pulmonary phthisis" may remain for ever unchanged.

It will be here observed that the term "Phthisis" is a generic one; for it is assumed that there are more kinds of consolidation than one; and it is contended that if there be consolidations differing in their nature, in their origin, in the signs and symptoms associated with them, in their relations to their environments, in their course and complications, in their duration and issues, it is a scientific duty to distinguish them; and it is, to say the least of it, a scientific retrogression to huddle them together under one name.

We are not unmindful of the fact that it is seriously contended by some persons of experience and eminence that, underlying the assemblage and progression of signs and symptoms to which we give the name of "Phthisis," there is but one form of pulmonary consolidation, and that, that form is invariably and truly tubercular. We cannot believe that this conclusion has been formulated in the light of any sound and extensive clinical experience; and even in the narrower, and sometimes misleading, light of pathological anatomy, we fail to discover by what facts and reasonings, a judgment could be established in such conflict with common experience, and doing such violence to the canons of scientific procedure.

It is demonstrated that, whilst the majority of pulmonary consolidations proceeding to excavation contain tubercular bacilli, and are therefore tubercular in nature, there is a very considerable minority which do not contain tubercular bacilli at any period of their history, and are therefore not tubercular in nature. Now, as a bacillary consolidation is radically different from a non-bacillary one, affects the organism in different ways, pursues a different course, contracts different relations, and ends in different issues, the necessities of empirical treatment, as well as loyalty to scientific method, require that they should be furnished with distinctive names. But even setting aside the anatomical contention, and, for the sake of argument, admitting as correct all that has been said in favour of unity in the nature of the consolidation, and all that has been said against variety, it does not of a surety follow that uniformity of structure postulates unity of nature in the processes producing it. For the true criterion of the nature of a disease, as we have said elsewhere, is not to be found in the anatomical forms through which it finds only a partial expression (and even that often shared by other affections); on the contrary, it lies in the complete life-history of the disease—in the continued grouping, and in the uniformity of course, relations, and issues of those dynamic states which precede, underlie, determine, and control those structural forms through which the true disease finds an incomplete expression.

A critical study of cases of chronic lung disease will easily enable an ordinary observer to divide them into two groups, one very large, and the other very small; and when the signs and symptoms characteristic of the cases composing each group are analysed, tabulated, and compared, and when the completed life-history of the one group is contrasted with the completed life-history of the other, it will be seen that the two groups are radically different throughout their respective courses, and that, according to the logical canons of terminology, each demands a distinctive appellation.

The strength of this argument will be more fully realised by the reader, if we place side by side before him the most prominent facts in the life-history of the two groups of cases to which we have been adverting:

THE LARGER GROUP.	THE SMALLER GROUP.
Origin insidious and seemingly constitutional.	Origin in some local inflammation, traumatic injury or specific fever.
Fever more or less continuous from first to last.	Except at the beginning, rarely fever throughout its whole course.
Features of subject delicately moulded; manners refined; nervous.	Features of subject full or heavy; manners unrefined; not nervous.
Body spare; fingers slender and pointed.	Body seldom spare; fingers clumsy and sometimes clubbed.
Heart small, quick, frequent, weak, and rarely much displaced.	Heart of full size, often enlarged, characteristically slow, and usually much displaced.
Cough frequent, hacking, percussive, rarely paroxysmal, unaccompanied by vomiting; occurs indifferently throughout the day.	Cough infrequent, usually paroxysmal and often accompanied by vomiting, sometimes by vertigo; occurs mostly in the morning.
Sputum usually purulent, clotted, nummular, discrete but variable; always contains tubercular bacilli.	Sputum commonly watery or frothy, or muco-purulent and diffluent; rarely discrete; sometimes fœtid; never contains tubercular bacilli.
Chest long, flat, narrow, and seldom very unsymmetrical.	Chest usually broad and deep, variously retracted and unsymmetrical.
Urine seldom albuminous.	Urine almost always, at some time, albuminous.
Secondary affections commonly tubercular.	Secondary affections commonly congestive or hyperplastic.
Recurring sensations of illness and exhaustion. Unequal to the ordinary duties and enjoyments of life.	Usually continuous good health and strength; unless exceptionally and towards the end, equal to the ordinary duties and enjoyments of life.
Lasts from two to five years, often hereditary.	Lasts from five to fifty years; seldom, if ever, hereditary.

The consolidations in the lungs of the patients constituting the larger group of cases are composed invariably of tubercular bacilli, of "tubercles," and of the various other structural changes which they bring about.

The consolidations in the lungs of the patients forming the smaller group of cases are composed almost entirely of fibroid tissue of different grades of organisation, and of some other inconstant structural changes brought about by derangements and interruptions of the local currents of blood and lymph.

To both groups the term "Phthisis" may be applied, if we

accept the definition of that state set forth in the beginning of this chapter. But in the larger group the chief agency in the production of phthisis is the bacillus tuberculosis; whilst in the smaller group the phthisis is dependent upon a fibroid transformation; tubercle playing no part in its production. To the larger group the term "Tubercular Phthisis" may be given; to the smaller, "Fibroid Phthisis." From these considerations, therefore, we feel that we are justified in retaining the term Fibroid Phthisis as denoting a disease, the main phenomena of which are indicated in the smaller group mentioned above.

CHAPTER III.

PATHOLOGICAL ANATOMY.

Morbid Anatomy of Lungs.

In this chapter we propose to deal with the pathology of pure fibroid disease only, leaving what we have to say about the pathology of the tuberculo-fibroid and fibro-tubercular varieties until we come to speak about these subjects.

It will have been gathered from the historical account, that although authors have differed from one another as to the anatomical situation of the morbid process, yet, when all their views are collated, there are really but three starting-places for this disease in the lung. According to some it may start from the pleura, according to others from the interlobular connective tissue, surrounding the bronchi and vessels; while yet others assert that it starts in the alveoli themselves. Could one but see this process at its beginning, it would not be difficult to say which of these situations was most frequently the site of the change; but when these cases come to the *post-mortem* table, the disease has, almost without exception, lasted a large number of years. The lung is completely invaded by an overgrowth of fibrous tissue, and it becomes impossible, except occasionally, to say where the starting-point of the process was. Authors may have assigned a situation as the most frequent place of origin of the disease, because they happened to have met with a large number of cases bearing out their particular contention. Thus, some have recorded cases arising from pleurisy; some arising from long-continued bronchitis, in many cases set up by the inhalation of irritating particles, such as stone-dust and coal-dust; whilst others have principally met with cases which have arisen out of an unresolved pneumonia. In each of these three respective instances, the inference is natural that the starting-point should be set down to the pleura, the tissue around the bronchi, or the alveoli themselves. So that while for the purposes of a systematic anatomical classification these three situations may

be admitted severally as the starting-point of the disease, yet it must be remembered that, when we have an opportunity for examining the lung, there is often nothing to tell us in what precise site the morbid process first began: all three of the localities being invaded to an almost equal extent.

In describing the morbid anatomy of this disease, it will be well first to give a systematic account of the results of the autopsies in general, and then to narrate the actual changes in each particular case, pointing out as we proceed, wherein we differ from the descriptions given by other workers in this subject.

The rarity of opportunities for making autopsies in these cases is perhaps one reason for the great want of agreement to be observed in the results of *post-mortems* already published. An observer may quite easily go for a number of years without being fortunate enough to obtain an examination of the viscera after death. It is often the case that these patients, after attending for years at a hospital, and finding themselves no better and no worse, at last cease coming altogether, and then by accident, the fact is learnt that they have died some time back. But although the number of autopsies is small, yet, when the infrequency of the disease is taken into account, it must be admitted that there is a sufficient number of them on record to allow of a fairly definite pathological picture of the disease, as seen *post-mortem*, being drawn.

Conformation of the Chest.—The general conformation of the chests of people who have died of fibroid disease is good, if we except the extreme contraction of one side, which is almost always present. There is, as a rule, found a thick covering of muscle, and often a fair quantity of subcutaneous fat. The cartilaginous portions of the ribs, especially those of the first three, are generally calcified, and require to be cut through with bone forceps.

Appearances on removing the Sternum.—On removing the sternum, instead of the heart and anterior mediastinum coming at once into view, the anterior edge of the least affected lung, immensely hypertrophied and very emphysematous, can be seen occupying and stretching across the anterior mediastinal space. The heart is altogether lost sight of, being drawn away and displaced along with the mediastinal tissues, towards the diseased side. It may here be noted that the heart is not simply twisted on its axis and drawn to one side or the other, but the anterior mediastinum as a whole is drawn over to the affected side. The hypertrophy of the least

affected lung is so extreme in some cases, that its anterior edge not only reaches across the mediastinum, but extends far into the opposite side of the chest. Indeed, one case is quoted in which, in addition to other signs, there was evidence of a cavity at about the third or fourth rib on the left side, about an inch away from the sternum. At the autopsy, it was found that this cavity was in the anterior edge of the otherwise sound right lung, which had extended over so as to take the place of the contracted one. At times the anterior edge of the least affected lung is found to extend only a few lines across the mid-sternal line; but there can almost invariably be noticed some encroachment of the least affected lung upon the mediastinum. The diseased lung is found occupying the posterior part of the cavity of the chest, and lying more or less towards the spine. The heart is bound tightly to the lung by firm fibrous adhesions uniting the pleura and external surface of the pericardium together. Sometimes the adhesions are so old and close that the lung has literally to be "cut out."

The Pleura, in cases of fibroid disease, may be said to be thickened to a greater or less extent in all cases. Most descriptions picture the pleura as being enormously thickened even to the extent of half or three-quarters of an inch. This may undoubtedly be so in some instances, especially in that form known as cirrhosis; but in many cases the pleura, though increased in thickness, is not markedly so, and in some others it appears to be almost unaffected. The extent of pleural thickening seems to bear no relation to the duration of the disease, for in two examples which have come under our notice, although the cases had lasted a long time, yet the pleura was only increased to about one-eighth of an inch. Altogether, judging from the cases which we have observed, the pleura is not nearly so thick as in cases of chronic tuberculosis of the apex, where it is no uncommon thing to find it measuring from a quarter to a half inch or even more in thickness. Whether the thickness of the pleura affords any help in the solution of the question as to the starting-point of the disease, cannot be determined; it would, however, be reasonable to suppose that if the disease started in that situation, the pleura might show evidence of involvement to a greater degree than other parts of the lung. However that may be, the pleura is almost universally thickened, and if one part of it is thicker than another, it is that which lies over the area of most extensive disease in the lung. On the cut

PLATE I.

This specimen shows well the thickened pleura. At the apex may be seen a cyst-like space in the cut surface of the pleural membrane, oval and flattened in shape, with smooth lining.

The cut section of the lung shows the fibroid material gathered together towards the root, and more sparsely scattered as the periphery is reached.

PLATE I

This specimen shows well the thickened pleura. At the apex may be seen a cyst-like space in the cut surface of the pleural membrane, oval and flattened in shape, with smooth lining.

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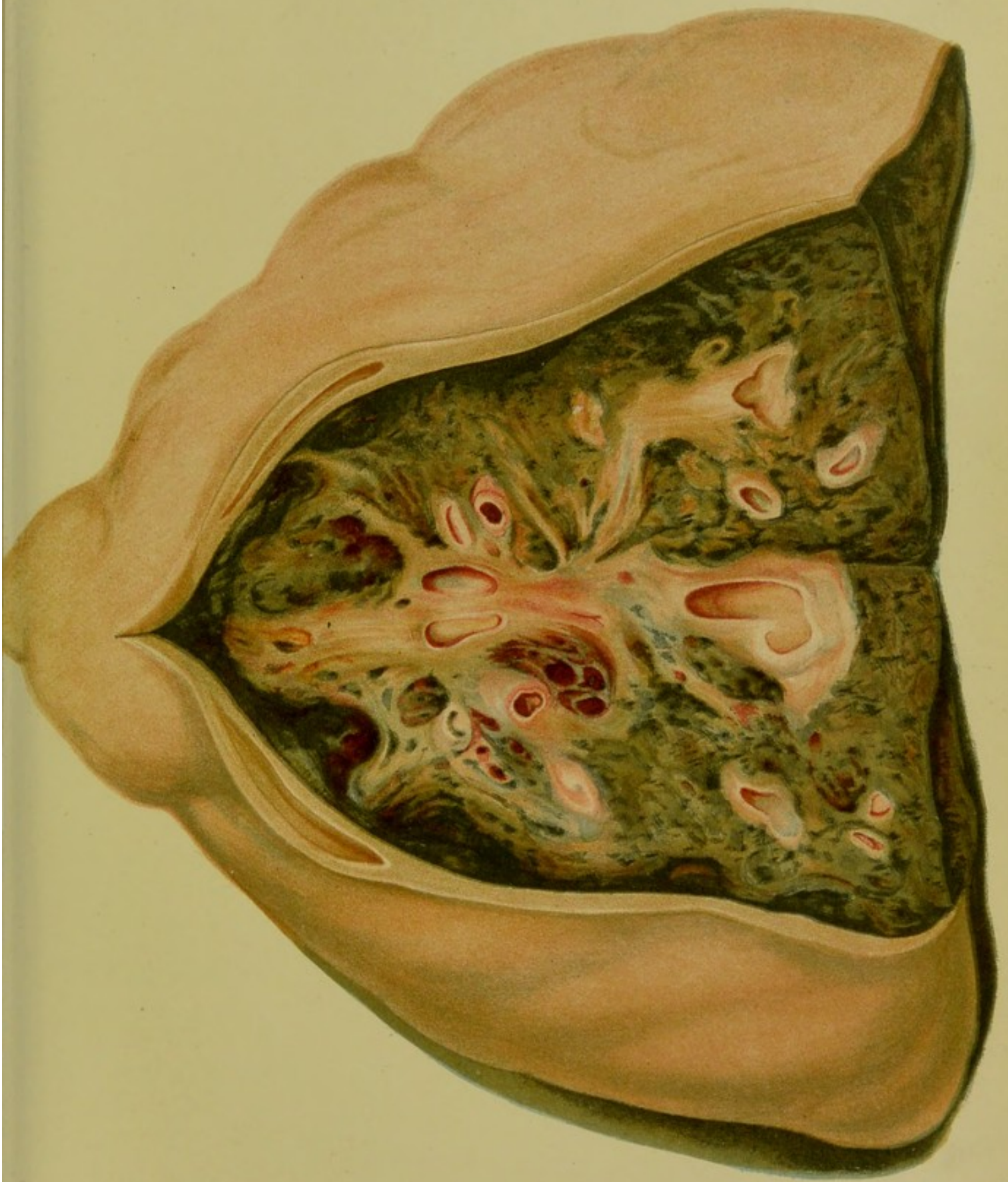


surface, the interlobular bands, much increased, can be seen dipping down into the lung from the deeper layer of the visceral pleura; giving the appearance of thin, white lines.

The **Pleural Cavity** is generally obliterated by tough fibrous adhesions, binding the two surfaces together. If the whole lung be diseased, the whole cavity is obliterated; if only a localised area is affected, usually that portion of the pleural cavity opposite the disease is closed up; but the cavity is sometimes entirely effaced without the whole lung being fibroid. Sometimes on section of the pleura there are seen cyst-like spaces, the walls of which are smooth. The origin of these is doubtful, but probably they are caused by the irregular way in which the bands of fibrous tissue become attached to the pleural surfaces; some adhering closely; some leaving loops and spaces, and enclosing in their meshes masses of soft lymph, which have become imperfectly organised. This condition of things is well shown in Dr. Coats' *Pathology*.

Size of the Lung.—The size of the lung varies considerably. In some cases its dimensions may be slightly decreased only; but in others it may be contracted to the size of the closed fist. In other instances of partial involvement of the lung by fibroid disease, that part affected (whether it be a lobe, or a part of a lobe) is contracted and shrivelled to a very small size, while the rest of the lung fills up the whole of the pleural cavity, its size having increased by compensatory emphysema. Why, at times, there should be great diminution of size, and at others scarcely any, is not at all clear. Duration of the disease will not offer a sufficient explanation. For, in one case coming under our notice, where the disease had lasted fifteen years, there was little or no departure from the normal size of the lung; while in another, where the disease had lasted just as long, the lung was no larger than the two fists. The solution of this problem must be sought for in the conditions under which the disease arises. The real cause of the contraction in the bulk of the lung depends upon the amount of contracting fibroid tissue therein. The wasting of the lung might possibly be rendered more easy by the diminished resistance due to softening of the chest wall, during the time the process was most active. If contraction of the lung set in and proceeded in early life, when the thoracic parietes were easily capable of yielding, it is probable that there would be a greater chance of the

lung becoming diminished in size, than if the process began later in life, when the thorax was firm and fixed. But whether the cases coming under observation bear out this suggestion is difficult to say; to speak with any degree of certainty would require a very long and patient investigation of this point.

In infinitely rare cases the fibroid change appears to be almost exclusively confined to the interlobular spaces, and in such cases, when the surface of a section of the lung is examined, it is seen to be much contracted, and to present the appearances of a true cirrhosis. A case of this kind is, or was recently, to be found in the Museum of the Royal Naval Hospital at Haslar.

The amount of compensatory emphysema in the lung is another point upon which its size depends. Where the disease is partial, emphysema may well take place, so that little diminution ensues. The position of the greatest amount of fibrous tissue also affects the contraction of the lung. For the purposes of the production of a decrease in the size of the lung, fibroid tissue is more effective if it is placed mostly in the interstitial tissue around the bronchi and vessels, and in the pleura, than if it is found in greatest excess in the alveoli themselves. This being so, one would suppose that the lungs of those cases which arise out of an attack of pneumonia would show less evidence of withering than those which had their origin in bronchitis and pleurisy. To this proposition, however, a positive answer cannot be given. Lastly, the situation of the disease might affect the resulting contraction. Disease more or less localised to the apex would produce less contraction than disease localised at the base, or affecting the whole lung. Be that as it may, it is important to bear in mind, that extreme diminution in the size of the lung is not an invariable concomitant of fibroid disease. That the lung is in all cases smaller than natural cannot be doubted; but for it to reach a size no larger than the closed fist, is a condition which, from our own observations, would seem to be of great rarity.

Consistence.—A fibroid lung is firm, tough, dense, and leathery to the touch; and in consistence resembles india-rubber. When pressed tightly between the fingers, the organ retains its shape and does not pit on pressure, unless emphysema and œdema be present. Throughout the lung can be felt lumps of harder texture than the surrounding tissue, which on section will be found to be dilated tubes, masses of fibroid tissue, or caseous nodules. The normal

external conformation of the lung is not greatly altered, but the division of the viscus into lobes is lost; the interlobar fissures being bound together, adherent, and completely obliterated by the results of old pleural inflammation and thickening. The density of the lung is much increased, and small portions of it, where its texture is of the closest consistence, sink in water. At other parts, where the process is not so complete, there is enough sound pulmonary tissue to allow of its floating.

On cutting into the organ, the tissue resists the knife, and, in some instances, a distinctly cartilaginous and grating sensation is obtained. The appearances seen on the cut surface depend very much upon the extent of the disease. If the fibrosis be very advanced, the lung seems to be made up almost entirely of fibrous tissue, dilated bronchi, and thickened vessels; the cut ends of which stand out prominently, all lung-tissue having disappeared. If not so advanced, the lung has the appearance of being intersected in all directions by white fibrous bands, dividing its tissue up into lobules and marking it out into planes. If carefully inspected, the fibrous tissue will be found in largest amount around the bronchi; and it can be followed up to the root of the lung, where often all the elements forming it are encased in fibrous tissue. Frequently, in parts of the lung not greatly affected with fibroid disease, a considerable amount of emphysema can be seen. Often the edges of the lung contain bullæ of large size. This emphysema is in the truest sense of the word compensatory, and is found only in those parts of the lung where the intimate pulmonary tissue is not already destroyed by the overgrowth of fibrous tissue. Emphysema of this kind never seems to attain large dimensions, and cannot for a moment be compared with the enormous hypertrophy which is the rule, rather than otherwise, in the least affected lung.

Compensatory emphysema in these cases is often a genuine hypertrophy, without atrophy in any form whatever. True emphysema, when present, may often be caused by obstruction of the blood-vessels; for this condition of true emphysema has been experimentally produced by one of us, by the injection of minute particles into the pulmonary vessel.

Colour.—The colour of the lung, when cut into, is a matter of considerable moment. Authors, as a rule, have spoken of the lung in this state as being deeply pigmented. The black colour-

ing matter is said to be most in evidence beneath the pleura, in the interlobular spaces, and in the bronchial glands; in other words, thickest in the regions occupied by the lymph channels of the lung and their appendages. They go on to describe the lung as looking quite black in extreme cases, and exuding on pressure a black, inky juice, which stains the fingers. The colouring matter, they maintain, invades all portions of the organ, and presents a uniformly black appearance. In other cases, where there is only a slight amount of pigment, it is described as having the appearance of dotted lines scattered all over the lung in the situations mentioned above. This is a perfectly appropriate description of the colour in very many cases of fibroid disease, but it does not by any means apply to all cases without exception. The mere fact of fibroid disease being present is not sufficient reason to account for pigmentation. One must seek in the antecedents of the case for the cause of the dark colour of the lung. A man who has spent his life in a coal-mine, and who has fallen a victim to fibroid disease brought on by long-standing bronchitis, will almost certainly have his lungs blackened extensively by pigment; and this concomitant is due to the accident of his surroundings. But another case of fibroid disease, which has begun after an attack of pneumonia or broncho-pneumonia, and which has lived far away from any influences likely to produce pigmentation, will, at the autopsy, present lungs which, though quite as unmistakably fibroid as the other, yet contain hardly more colouring matter than normal. Of course, the fact that the lymphatics are blocked and obliterated by inflammation, renders it impossible for the dust in the inspired air to pass through and become absorbed.

Some lungs affected with fibroid disease contain even less pigment than normal. The pulmonary substance has a greyish hue, and in some places is almost white. This light colour is produced by the large amount of white fibrous tissue closely packed together, and the absence of blood. When more sparsely scattered, the fibroid change appears as thin white lines, as has been before mentioned. Sometimes again, the lung has a slaty-grey appearance. In one case we examined, the organ in its anterior portion was of a light buff colour, in the posterior and basal portions slaty-grey, and all the pigment that could be seen consisted of a few spots, distributed evenly all over the lung.

The **Bronchial Glands** are always enlarged to a varying extent. They may be hard, and covered by a thick fibrous envelope; on section they may be seen to be overrun with fibrous growth, and here and there may be demonstrated pigmentation. Nevertheless, fibroid changes in the glands, as far as our autopsies go, are the exception rather than the rule. It is rare, according to our experience, to find caseating centres in the glands. The fibrous overgrowth spreads out in the form of white lines intersecting the gland, or is seen as circumscribed masses. The bronchial glands are not, perhaps, so large as in cases of tuberculosis, and, so far as has been ascertained, they do not appear to be softened and œdematous, as is so often observed in bronchial glands enlarged as the result of tuberculous deposit.

The **position of the lesion in the lung** has been variously described by preceding authors. Some have spoken of the apex as being the commonest situation for this affection, others have just as precisely indicated the base as being the most frequent site for fibroid disease, while in many instances no special locality of the lung has been claimed as being most liable to it. Now, although it cannot be said that fibroid disease favours invariably any particular part of the lung, yet, from the cases we have collected, and the *post-mortem* examinations we have been able to perform, great support is given to the belief that the middle and base of the lung are the common seats of this disease. Indeed, it is not too much to say that fibroid disease affects the middle and base just as constantly as tubercular disease does the apex, and that those cases in which the summit of the lung is primarily attacked, must be taken rather as exceptions which prove the rule. Even when the whole lung is overrun with fibroid tissue, the process is most advanced at the middle and base, and whereas in a tuberculous lung all gradations may be traced from the old, dry, cavity at the apex, to the recent fresh miliary granulations at the base; so here one may make out the complete fibroid metamorphosis at the lower parts, passing upwards in successive stages, until at the apex there is found newly formed fibrous tissue, or healthy lung. Again, when the disease passes over to the other lung, it is not the apex which is first attacked, but generally the basal parts. These opinions are borne out by many observers, but it should be mentioned that Sutton, Rokitansky, and Laennec held that the common

seat of the disease was the apex. So much are their opinions at variance with the generally accepted views, that it might almost be thought they were dealing with cases of chronic tuberculosis, in which the fibroid formation predominated.*

As seen by the naked eye, two forms of fibroid disease may be distinguished.—**a diffused form and a circumscribed form.** The fibroid tissue may be more or less evenly diffused over the whole lung or parts of the lung, or it may appear as well-defined masses varying from the size of a pea to that of a filbert. These masses are hard, and consist of fibroid tissue. Sometimes, though rarely, they have small spots of softening in their centres. The intervening lung-tissue shows increase of fibroid material. By some authors this condition is termed nodular cirrhosis, and is thought to be really aggregations of tuberculous matter, which have taken on a fibroid change; in this form the bronchial tubes do not seem to be much dilated.† But the diffused form is the common one, and presents appearances such as have been described in this chapter.

Cavities and Caseous Deposits.—At times, though rarely, one meets with cavities and small areas of caseous softening, in addition to the appearances already described. We do not mean to include, in the term cavity, those vomicæ which are formed by the dilatation of a bronchial tube, but simply those which are produced by the softening and liquefaction of deposits, and those rare cases in which the walls of the bronchial dilatation have become completely destroyed, and have resulted in the formation of a cavity.

A discussion as to the true nature of these cavities and caseous deposits is of great importance, for whatever conclusion is arrived at will go far towards deciding the question of the dependence of this condition of fibrosis upon tuberculosis. The presence of excavation is by no means constant in fibroid lungs. In three cases that have come under our notice, although the lung was cut in all directions, yet in one of them not a vestige of a cavity could be found; and in the other two, at last, after a diligent search, a small cavity in each was observed, about the size of a bean, containing caseous matter.

* In consequence of this distribution a rule may almost be formulated that contraction in the lung extends from the periphery towards the root.

† Ziegler, *Pathological Anatomy*.

There is a cavity—narrow, sinuous, and branched, irregular, imperfectly lined, and bloody—which is usually quite free from caseous matter. Such cavities sometimes take their origin in small bronchial tubes by ulceration spreading along them, which destroys the bronchial walls and lining substance. Now there can be no doubt that the majority of writers have admitted the existence of breaking down and caseation in fibroid lungs. Some of them attributed it to ulceration, others to gangrene, while yet others were describing that form of fibroid disease which is sometimes found associated with chronic tuberculosis. To this last category, in strictness, should be relegated most of the cases given by Dr. Sutton in his well-known article. When caseous deposits and cavities are found in lungs which, in their origin and cause, are purely fibroid, and bear no relation to tubercle, they are generally of small size, and situated in the densest parts of the fibroid material. They are not plentiful in number, and bear no proportion to the amount of fibroid tissue in the lung, and seem to depend for their origin upon the starvation of the fibroid material of its blood, and consequent necrosis, rather than upon the deposition of tubercle. Another point worthy of notice is, that these deposits and cavities are not usually found at the apex of the lung. It cannot be said that fibroid tissue in the lung is certain to end in caseation, for it is a matter of common experience to find slight caseation in one dense patch of fibrosis, while in other patches near by, and just as old and quite as dense, there is no evidence of necrosis to be seen.

There is no naked-eye appearance of caseous deposits and cavities to call for special notice; the important question of the possibility of a tubercular origin of these conditions will with advantage be left to be discussed when we come to speak of the microscopical characters of the lung; suffice it to say at present, that because, in a fibroid lung, there is caseation or excavation, it must not therefore be too quickly assumed that the cause of these conditions is tubercle, and still less that the whole fibroid process is due to them.

It should have been said, while speaking of the difference between the tuberculous and non-tuberculous cavity, that the wall of the former has a distinctly membraniform character, while the latter has no such appearance.

Bronchiectasis.—We must now consider the state of the bronchi in pure fibroid disease of the lung. Bronchiectasis is,

perhaps, the most constant accompaniment of the affection. It is true that a few authors have spoken of the bronchial tubes as being little, if at all, dilated. The opinions of these authorities may possibly be accounted for on the supposition that the cases on which they based their assertions were not advanced, and consequently presented no great change in the bronchi. So prominent a feature of fibroid disease is bronchiectasis, that before the time of Corrigan it was commonly believed that the tube dilatation was primary, and the fibrosis secondary. It would be indeed difficult to meet with a fibroid lung in which there was no bronchiectasis, and, with the few exceptions mentioned above, all are agreed as to the enlargement of the calibre of the tubes taking place in fibroid disease. In this matter, our experience certainly coincides with the majority.

In fibroid disease, there is always evidence of chronic inflammation of the bronchi. The mucous membrane is of a dull red colour, and deeply congested; sometimes rough and uneven, sometimes smooth and shining, sometimes ulcerated. Very often it is thrown into folds and creases.

When the lung is pressed between the fingers, large quantities of yellow muco-purulent secretion well up from the bronchial tubes. As a result of the chronic inflammation, the wall of the tube becomes thickened. When the bronchiectasis is at all extensive, this thickening is not always apparent; but on comparing the tube with those of the sound lung, it can almost always be demonstrated that its wall is thicker than usual.

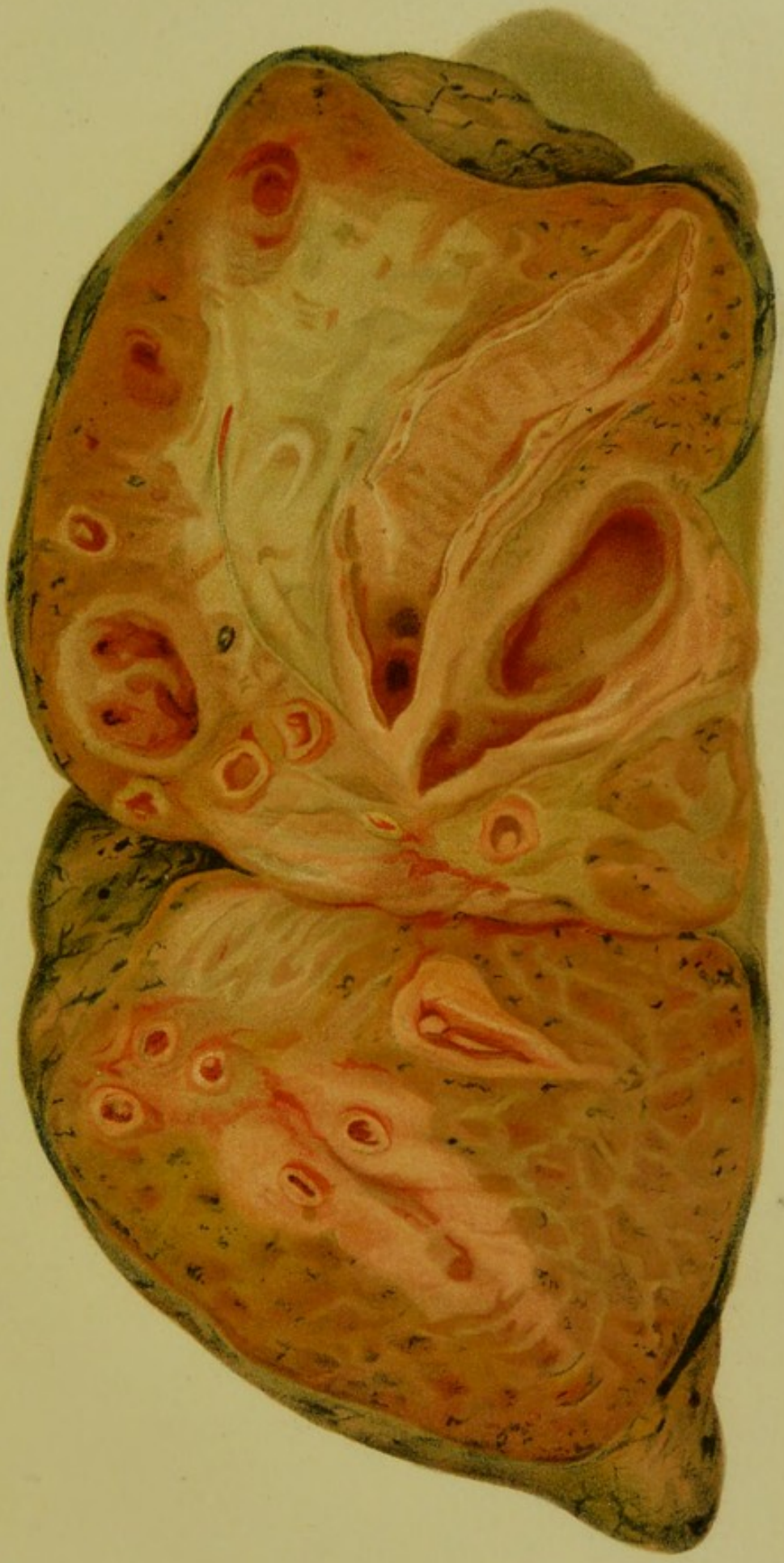
When the main bronchus belonging to the affected lung is cut across as it enters the organ, an increase of thickness in the wall can as a rule be easily noticed; and sometimes a slight increase in the calibre of the tube can be detected, as soon as it has fairly entered the lung. This latter appearance is, however, not often seen; but it may be mentioned that in one of the cases coming under our observation, on slitting up the bronchus, an oval-shaped dilatation was found just at the spot where the lobar branches are given off.

Bronchiectases are generally described as being either cylindrical or sacculated; and it is true that most specimens of fibroid lungs show this to be the case. But there are other kinds which may be regarded as subdivisions of these two main forms. We might, indeed, with advantage describe the different varieties of bronchiectasis occurring in fibroid disease of the lung in the following way.

PLATE II.

This drawing is a good example of a fibroid lung, with dilated bronchial tubes. To the right is seen a long, irregular, dilated bronchial tube, with thickened walls. Two large (presumably bronchial) dilatations are seen in the right half of the lung, and many smaller ones scattered all over.

The lighter shading of the drawing shows the most advanced condition of fibroid degeneration. The darker represents what has been described by Addison as "buff-coloured," or total albuminoid induration. The pleura in this lung is not markedly thickened.



1. **The Cylindrical Form**, in which the bronchial tube is uniformily dilated throughout its whole diameter. When such a tube is slit up the dilatation is found to extend to the ending of the tube beneath the pleura. Sometimes in this form the dilatation is insignificant, the calibre of the tubes being but slightly increased; and it is important to notice that when this is the case, the walls of the bronchial tube are very thick, and stand out upon the cut surface of the lung. Perhaps this unusual increase in the thickness of the walls explains why it is that there is so little dilatation. The large amount of fibroid elements around the bronchi, and the hypertrophy of the walls themselves, might be supposed to offer a resistance to the dilating effects of expiratory pressure. But, on the other hand, it must be added, that if one accepts the view of Corrigan as to the cause of bronchiectasis (which assigns it to the contractile forces of the fibroid tissue gradually drawing the tubes apart), this increase of fibroid tissue around the bronchi would assist rather than retard their dilatation. There is another variety of the cylindrical dilatation in which the calibre of the tubes is increased to many times its natural size, and the walls are only slightly hypertrophied. Indeed, some say that the walls are even thinner than normal. We believe, however, that there is almost always to be found some increase of thickness. In this variety, on cutting into the lung, large, smooth-walled openings are found, sometimes admitting the little finger. These dilated tubes take a fairly straight course from the root of the lung to the pleura. The usual and not inappropriate description of these dilatations is that they resemble the fingers of a glove dipping down into the lung.

2. **The saccular variety of bronchiectasis** is also met with in fibroid disease; the common form of this variety is that in which, throughout the length of the tube, there are globular dilatations, the intervening parts of the bronchi being of normal size. When a fibroid lung with this condition of things is opened, the cut surface is found studded with small cup-shaped spaces with smooth shining walls, some as large as a bean, others no larger than a pea. The opening can generally be found at the bottom of the cup, which on dissection leads into another space of similar form. If the tube is dissected out, this moniliform arrangement is well seen. This condition has been well described by the late Dr. Fagge in his Text-Book.

It is rare to meet with a single bronchiectasis involving but one tube in fibroid disease, and equally rare is it to find bronchiectasis as the result of ulceration of the walls of the bronchi. As a rule, the bronchiectases are always multiple, and confined to that part of the lung in which the fibroid disease exists; and from this it follows that the common situation for bronchiectasis is the middle and lower parts of the lung. In connection with this it may be noticed that many have named the apices of the lung as the commonest site for bronchiectasis; but, as has just been stated, whatever part of the lung is most prone to fibrosis must also be most frequently the seat of the tube dilatations, and the common situation for fibroid disease is generally the middle and lower parts of the lung. To conclude the description of the gross morbid changes in a fibroid lung, there now only remains to speak of the vessels; and these may be dismissed in a very few words. The branch of the pulmonary artery going to the diseased lung is sometimes smaller than natural, sometimes not at all altered. On entering the lung many of its branches become obliterated, and look as if they had been ligatured from the main trunk, while other branches show evidence of hypertrophy of their walls.

Minute Changes.

In considering the microscopical appearances presented by the various fibroid conditions of the lung, it will be more satisfactory to take each tissue of that organ, and consider separately the changes occurring in it; the condition as a whole having been portrayed in the description of the coarse anatomy. It will be gathered from that description that the changes may be great or small in amount, that they may chiefly affect the interlobular tissue of the lung and those parts most intimately associated with it, or may find their chief seat in the alveoli themselves, and that the pleura may be greatly or but slightly affected. It is necessary to remember, therefore, that all the changes in the advanced form, about to be described, will not always be present in any one part; but that when any particular tissue of the organ is involved, it will present more or less the condition and appearances here given. Furthermore, it must be borne in mind that the lung as a whole, and especially that part of it in the neighbourhood of a fibroid mass, has its circulatory and other dynamic and static conditions

so profoundly disturbed, that none of its elementary anatomical constituents can escape structural alteration. It is these changes which, by their gradual growth and development, aided perhaps by slight, fresh inflammatory troubles (usually taking the form of patches of vesicular pneumonia, giving rise to groups of degenerative epithelial cells), lead to the further production of the disease. Whether the trouble originally began in one of the three tissues of the lung usually affected—viz., the pleura, the interlobular tissue and the parts it includes, or in the alveoli themselves—one tissue is so intimately associated with and related to another, that sooner or later each in its characteristic manner will tend to take part in the further progress of the disease. Thus, in the recent and more slightly affected lungs, we often find one or other tissue presenting more marked changes than the others, whereas, in the most extensive and long-standing cases, the whole of the various anatomical elements of the lung have almost always become the seat of disease more or less advanced. That tissue, however, which was first affected, is often much more so than those secondarily implicated, and gives a prevailing character to the whole lung, by which in many cases the disease may be recognised as one of pleuritic, interlobular, or alveolar origin. This difference, however, is infinitely more easily seen with the naked eye than under the microscope, which shows the changes in the individual tissues to be the same, whether they have been the result of a primary or secondary affection.

The description of the minute anatomy has been also confused and rendered unnecessarily complicated by separating into rigid classes, designated by different names, varieties which are all the result of the same process working in different tissues, in different stages and conditions, with a tendency to extend and perpetuate itself. This process is a chronic hyperplasia and inflammation of the various tissues of the lung. If the alveoli were the chief seat of this change, naturally we should expect some modification of the pneumonic process; if the interlobular tissue, an increase in amount with an alteration in character of this element. So also, if the pleura showed the most marked change, should we expect thickening and adhesion of that membrane, with a tendency to extend by thickened bands of scar-like tissue into the substance of the lung, with the interlobular tissue of which it is so intimately associated. Therefore, we find observers referring the condition

sometimes to "chronic pneumonia," sometimes to "interstitial pneumonia," whilst others have ascribed it to a "chronic pleurisy." It is not necessary to particularise further; but with regard to the term "interstitial pneumonia," if it is to be applied to the connective tisssue between the lobules, it is a rational enough designation; but if it is intended, as Williams and others intended it, to apply to an inflammation of the alleged connective tissue between the alveoli, the term is applied to a condition which can have no existence, and is irrational. For the wall of one alveolus is also the wall of the alveolus adjacent to it, and although this wall may be thickened in various degrees and ways, there can be no free exudation into any space between the alveoli, which space in fact does not exist. No doubt one or other of these three conditions was the initial cause, and might even remain the main factor in any individual case; but it is hardly possible to insist too strongly that in almost every instance, sooner or later in the disease, all these processes will be found to co-exist in varying proportions. Reasoning from analogous affections in other organs, this is exactly what one would expect. The kidney has been already instanced; and as in that organ we know how practically impossible it is to have an inflammation completely limited to the capsule, or the interstitial tissue, or the tubules, so also is it in the lung. Further, the more prolonged and chronic that affection proves, the more do we see the interstitial element creeping in, until, in kidney and lung alike, the last stage of long-continued disease is a contracted and fibroid condition, in which all the different tissues of the organ are more or less extensively implicated, and the functions of the whole more or less gravely impaired. The analogy ceases with the impairment of function, but in the case of the lung we have all the further changes, resulting from its contraction, occurring in the chest wall, the surrounding organs and vessels, in the air-tubes and lung-tissue, as well as in the lymphatic and blood-vessels contained in its substance. It is chiefly upon the signs and symptoms caused by these changes that we depend in making a diagnosis of the fibroid and contracted condition of a lung.

Bearing these facts in mind, we will proceed to discuss the anatomical changes met with in this condition in the various elementary anatomical constituents of the lung taken seriatim.

Pleura and Interlobular Tissue.—As we have seen, the

pleura is generally thickened, though usually not to the extent that is often found in tubercular cases, and sometimes, even when the disease is advanced in other parts, as may be seen in the case of A. K., this membrane may be found but slightly, if at all, affected. Microscopically, this thickened pleura can generally be defined as consisting of an outer and an inner layer. Both of these are largely composed of fibroid tissue, but the outer of the two consists almost exclusively of this material. The fibroid material in the **outer layer** shows great regularity of the fibres, which are arranged more or less parallel to the surface of the lung, well organised, containing for the most part but few new elements in the shape of round cells, and having in its meshes numerous dilated and thickened blood-vessels. The lymphatic channels in this layer are markedly dilated, so as to form quite an open network near the surface. Outside this layer may occasionally be seen a third, which can easily be shown to be a portion of the parietal pleura, stripped from the chest wall in removing the lung, to the visceral pleura of which it has become densely adherent. Between this and the outer layer may often be seen elongated spaces, lined by a single layer of flattened epithelium, showing the remains of the now obliterated pleural cavity.

The **inner layer,** like the outer, is largely composed of fibroid tissue, but in it the bundles of fibres show no regular arrangement, being curled and twisted in all directions. In its meshes are numerous small, round, nucleated cells; some are seen dividing, and others becoming elongated into oat-shapes and losing their nuclei. In most cases it is almost impossible to determine where the pleura ends and the altered and sclerosed lung-substance begins. Both alike are composed of irregularly disposed bundles of fibroid tissue, infiltrated with numerous small, round, nucleated cells, and containing, in addition to the thickened and dilated blood-vessels, seen in the outer layer, many newly formed capillary vessels filled with blood; but no traces of the alveoli are left in what was originally pulmonary tissue. By the addition of caustic potash, or acetic acid, the redundant connective tissue becomes expunged, and the elastic tissue of the former alveoli may be recognised. The network of new capillaries forms quite a feature in some parts, and so numerous are they occasionally, as almost to merit the term nævoid. Sometimes the margin between this fibroid envelope (which, as hinted above, is, in all probability, partly composed of

altered lung-tissue) and the more or less healthy pulmonary tissue beneath, is abruptly defined. At others, the two shade more gradually into one another, and always there may be seen numerous septa of sclerotic tissue dipping into the lung from the pleura, to join the usually thickened and condensed interlobular tissue. The lymphatic channels are not at all numerous in this layer, though occasionally connecting branches may be seen, passing from the large network in the outer layer, into the lung along the above-mentioned septa. Lastly, in most parts of the thickened envelope, granules of pigment are seen, sometimes lying in the interstices of the tissue, and at others apparently in blocked lymph spaces. These are much more numerous in those lungs in which the disease is the result of inhaled dust, but are present to some extent in all.

Interlobular Tissue.—Turning now to the consideration of the interlobular tissue, we find that in all advanced cases it is more or less increased in amount, and considerably condensed and fibroid in structure. It must be remembered that of the bands which are seen and felt traversing the affected portions of the lung, some undoubtedly arise from the increase and thickening of the original interlobular tissue of the organ. Others owe their chief origin to the pleura, as seen in the description of it above. There is yet another way in which these fibroid bands are formed, and that is by the condensation and conversion into fibroid tissue of the lung-substance itself; and perhaps, of the three ways, this accounts for the greatest part of the fibrosis. It must, however, be noted that these bands, although they may owe their chief support to the pleura or to the condensed lung-tissue, yet are usually formed round strands of pre-existing but altered interlobular tissue, and may generally be seen to contain the lymph- and blood-vessels of the part. Therefore it will be seen that the interlobular tissue plays a very important part in the process, although there are bands of fibroid material formed independently of it; and when large areas are affected, its increase is insignificant in comparison with the gross lesion. The formation of fibroid material from lung-substance will be dealt with in the consideration of the alveoli. The part played by the pleura in this change has been referred to. It remains, however, to study here the altered structure of the interlobular tissue, and the result of this change upon the vessels and lymphatics which it contains.

When a section including a portion of the altered interlobular tissue is examined microscopically, it usually presents the appearance of more or less well-organised fibroid material, containing in its meshes a few lymphoid cells; some large and thickened blood-vessels, some of which are quite obliterated; whilst the lymphatics are for the most part so altered as to be no longer recognisable. The earlier steps of this process may generally be traced in different parts of the lung, or gathered from the appearances presented in other lungs where the disease is not so advanced. In all inflammatory troubles of the lung, the interlobular tissue will be seen to take part, and the earliest changes may be well studied in pneumonia, and more especially in those cases which, from some cause or other, have failed to resolve for a considerable time after the usual period for resolution. In such a case, and often too in fibroid cases, in those areas which have been but recently involved, the following changes may be observed. Numerous lymphoid cells are seen in the meshes of the connective tissue of the part, some probably derived from the blood-vessels found there, and others from the connective-tissue cells which can be seen actively dividing. Where the neighbouring alveoli are also the seat of inflammation, crowds of these lymphoid cells may be seen invading the interlobular septa from them. Later, many of these new elements become more or less perfectly organised, the cells elongating, and finally losing their nuclei, so that the septum may be seen to be increased by a fibro-cellular growth; which later again, tends to become more or less completely fibroid; the cellular elements becoming less and less distinct. In the first stage, the capillaries, where seen, are found distended and tortuous; later, in the fibro-cellular stage, there is often a very considerable increase in their number so as to give the part great vascularity. Generally, at this stage, many of the neighbouring alveoli have become involved, and being often similarly vascular, large areas of almost nævoid tissue are sometimes seen, reminding one forcibly of the like appearance noted in the deeper layer of the pleura. This condition will be again referred to when speaking of the affection of the alveoli, where its importance in often giving rise to hæmoptysis will be discussed. (*Vide* Figs. 1, 2, 3.)

Finally, the stage first described of more or less well-organised fibroid tissue is reached, there being now but few vessels, and those large and thickened as to their walls. The capillaries have wholly

disappeared, often many even of the vessels proper to the part have become obliterated, and the whole structure tends to become more fibroid and less vascular and cellular. Lastly, there may be seen in this tissue, as elsewhere in the lung, pigment granules; this change, as before remarked, being more apparent in the lungs of those engaged in dusty employments. This would seem the best place to speak specially of the vessels and lymphatics, which present marked changes throughout the lung. Some of these changes have been mentioned, and others will be referred to later on; but as the minute appearances are similar in all, they will be here described. It will be seen from what has already been said, that the blood-vessels found in the diseased organ are: 1. Those originally belonging to it; 2. Those which have been newly formed.

1. **Blood-vessels proper to the Lung.**—Of these, let us first mention the larger ones. If the larger vessels be slit up from the root to the periphery of the organ, their walls will be found somewhat thickened, and in many cases presenting patches of atheroma; but this change is not marked, and calls for no special description. As one passes to the medium-sized and smaller vessels, one may observe that some have become wholly obliterated, whilst those that still remain pervious present considerable thickening of their walls, and, in some instances at least, are larger than normal. Taking first the impervious vessels, little need be said as to the minute changes. They may be traced often for some distance as fibrous cords, and then become quite lost in the dense fibroid tissue of the part. It is important to note, however, that the areas of lung which they formerly supplied are, on the one hand, converted into dense fibroid tissue; or, on the other, show well-marked emphysema. That is to say, these obliterated vessels are found largely where the fibroid change in the lung is so advanced that the whole, or indeed large areas, are converted into practically unused appendages of fibroid tissue. Where a vessel leading to a part, not yet involved in the fibroid change, becomes nipped and impervious as it courses through a diseased area, then it may be sometimes observed that the part drawing its supply from such a vessel has passed into a state of well-marked emphysema. Those vessels with thickened walls, but which were still pervious (and these formed by far the larger number), were found to owe the thickening, partly to some hypertrophy of the muscular layer, but mostly to a marked increase of the external coat, which

showed distinct fibro-cellular structure. No changes were noticed in the endothelium.

2. **The newly formed vessels** present no peculiarity worthy of note as to their structure. They are capillaries, and are found mostly in the deeper layer of the pleura, which, it will be remembered, was richly cellular, and in the new cellular growth of

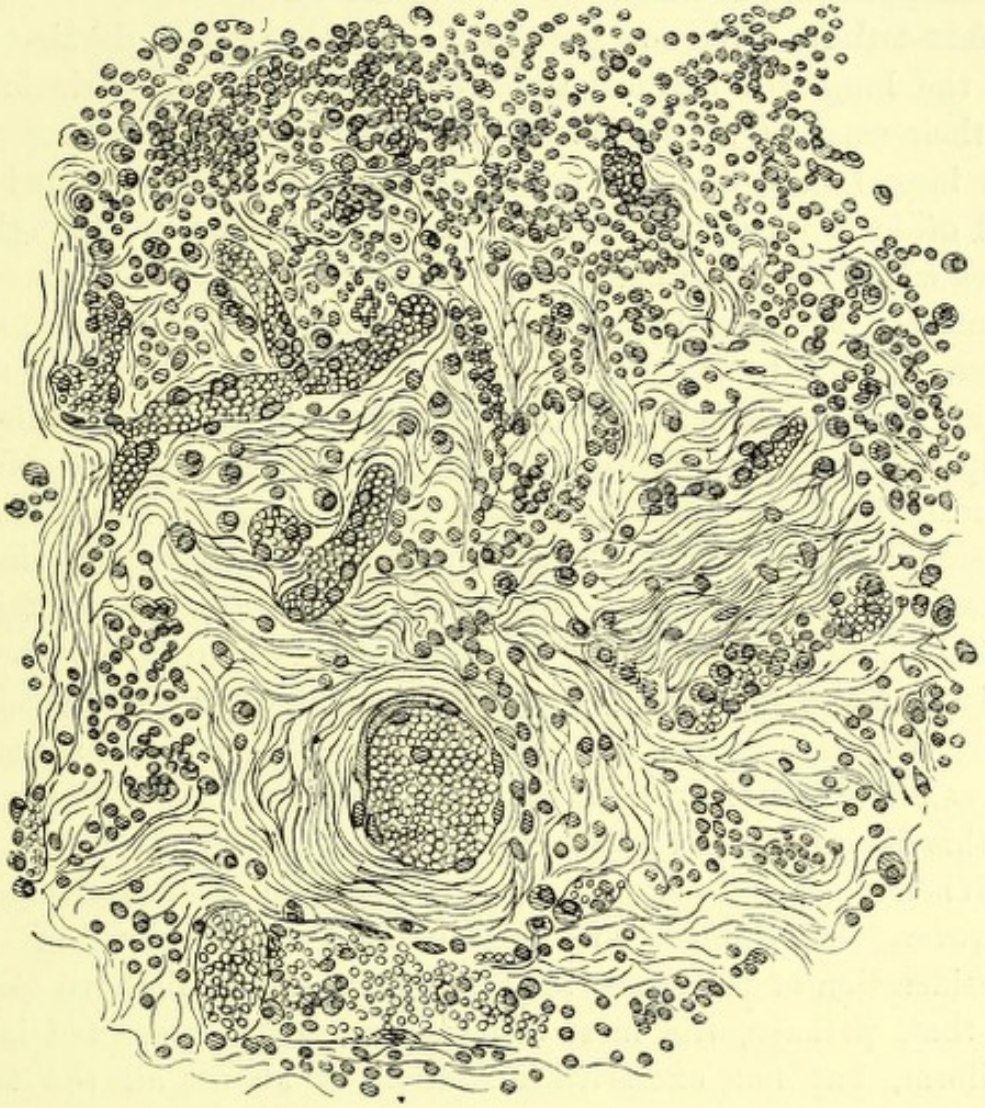


FIG. 1.—Showing extreme vascularity of the newly formed fibroid material, giving it more or less the appearance of granulation tissue; all trace of the alveolar walls being lost, and many new vessels being in process of formation.

the alveoli and interlobular tissue. Indeed, where the change is very active, these tortuous capillaries, imbedded in crowds of lymphoid cells, present exactly the appearance of granulation-tissue, and as this material becomes organised, so the vessels tend to disappear. (*Vide* Fig. 1.)

3. **The lymphatics**, except in the outer layer of the pleura, where they are considerably dilated, are early obliterated. Probably a large amount of the pigment-granules was originally in these channels, and in "*dust lungs*" the lymphatics, where still recognisable, are seen loaded with pigment and particles of the matter of which the dust is composed. The dilatation of the pleural lymphatics is easily explained by the blocking of the channels, as they course to the root of the lung.

Air-tubes.—We now pass to the consideration of the air-tubes of the lung, the trachea, and the bronchi, from their larger to their smallest ramifications. **In the trachea**, the changes are not important. We have not noticed any dilatation of this tube, and often it presents a perfectly normal appearance, though in cases where there has been long-continued bronchitis, the mucous membrane shows the changes which are ordinarily seen in such cases. That is to say, the cilia are shed, and, in some instances, many of the superficial cells of the mucous lining also; while the deeper layers show some proliferation, associated with fibro-cellular increase in the submucous layer. Small superficial erosions or ulcers may also occasionally be seen, and from these, and similar ones in the bronchi, small portions of elastic tissue are sometimes extruded, but never, so far as we know, presenting the characteristic areolar form of the alveoli. Beyond these slight changes, there is nothing worthy of note. The glands present a normal appearance, and the cartilages do not appear to calcify any earlier in this disease than in normal cases.

When we come to examine the **bronchi** and their ramifications, however, the changes are most marked and interesting. The consideration of the coarse appearances in them will have taught us that, perhaps, the most usual characteristic presented is the uniform, but not excessive dilatation of almost all the tubes throughout the affected lung, associated with more or less thickening of their walls. The minute anatomy of these thickened walls will first be considered. Then we shall describe the appearances presented by the walls in those cases where the tubes are dilated to many times their ordinary size, or expanded into globe-like cavities. Further, we must say a few words with reference to the condition of the mucous membrane throughout the tract, and lastly, consider the appearances and formation of cavities connected with the bronchial tubes.

If a section be made of the thickened wall of a tube, dilated to about the size of a goose's quill, the following changes may be observed microscopically. As in cases of bronchitis, the cilia are all shed. The columnar cells of the mucous lining are also, in most places, absent, while the deeper layers of cells have proliferated, and form a layer of irregularly shaped cells lying on a very considerably thickened basement membrane. In some cases, all the cells are shed down to this thickened basement membrane, while yet again, in others, this membrane itself may be indistinguishable from the deeper fibroid tissue; or the tube may be lined with granulation tissue, no trace of the mucous lining or basement membrane being left. The submucous tissue beneath the basement membrane is generally found considerably thickened by a fibro-nuclear overgrowth, which, on the one hand, may be rich in cells, and contain many new capillary vessels; or, on the other, may be more or less perfectly organised into fibroid tissue, with a considerable diminution in its vascularity and cellular structure. The glands of the bronchial tubes are sometimes plainly visible, and seem but little altered, whilst at others, they may be seen imbedded in fibroid tissue, and undergoing atrophy; and in many cases are altogether wanting. The cartilages withstand the changes for a long time, but many of them eventually atrophy and disappear; and this process may be traced in different sections, in some of which they appear normal; in others their outline has become irregular and shreddy, and the cells are fast disappearing; whilst in yet others, they are wholly absent. Outside them is generally seen a dense layer of fibroid tissue, forming by far the greater part of the whole of the thickened wall of the tube. Indeed, not unfrequently, nearly the whole of the tissues inside the cartilages have disappeared, and the tube wall is formed by these two constituents only, whilst in a few cases the wall is composed alone of fibro-cellular tissue, no trace remaining of mucous membrane, glands, or cartilages. In these last, it is naturally difficult to say what the origin of this tissue was; whether any of the original tissues of the tube, now converted into fibroid material, went to make up part of this wall, or whether they had all been shed, and the substance remaining was simply the product of fibroid condensation of the surrounding interlobular and pulmonary tissues. However that may be, the result is the same; the walls are entirely composed of fibro-cellular tissue, and none of the original

anatomical constituents can be recognised. In those cases where the bronchial tube is dilated to many times its normal size, much the same appearances as those described above may often be observed. But here sometimes the wall of the tube is thinner than normal. This thinning of the wall was not remarked in the large or medium-sized tubes; but where the dilatations affected the smaller bronchioles, and it seemed to be most commonly close to where the smallest ramifications opened into emphysematous lung. The condition of the mucous lining at various parts of the tubes requires some special notice. We have seen that the cilia are always shed; further, that often the columnar cells are wanting, or the whole of it down to the basement membrane may be absent. Sometimes the mucous membrane presented to the naked eye a velvety appearance. Microscopically, this was found to be due to the fact that, the lining was composed practically of granulation tissue, presenting more or less distinct papillæ, composed of lymphoid tissue, each with its own tiny capillary vessel. Here the whole of the innermost cells of the mucous membrane had been shed, and the granulation-tissue was formed from the submucous layer. It was, as will be gathered, very vascular, and would no doubt often be responsible for more or less serious hæmoptysis. In some of the larger tubes, which were at the same time dilated, a fenestrated appearance of the lining membrane was observed. When a section was made of such a portion, it was found that the submucous tissue had thickened into tiny, irregularly arranged bands, over which the mucous membrane was stretched, and between which it had sunk, so as to form little sacs or pits when looked at from the lumen of the tube.

Now with regard to ulceration of the tubes.—No doubt the condition described above, where the lining membrane is composed of granulation tissue, is, strictly speaking, one of superficial ulceration; but in rare instances this process extends deeply, so as to involve the whole of the constituents of the wall; and as in such cases it often extends along the tube and its ramifications, extensive, ragged-walled, dendritic or branching ulcers are thus formed. So it will be seen that the ulcerative process may be but a superficial erosion, or may extend completely through the wall of the tube, and even invade the pulmonary tissue beyond. This brings us naturally to the consideration of the cavities in connection with the bronchial tubes. The above description will

serve to show how they may sometimes be formed by extension of ulceration from a tube in a more or less deep and circumscribed direction, or may be, as it were, simply a certain length of ulcerated tube; in the former case being more or less regular in shape, in the latter, irregular and branched. The walls of such cavities will be found ragged, often sloughy, and limited by altered pulmonary tissue, or by the remains of the walls of the tube from which they originated. Sometimes smooth-walled cavities are found communicating with the bronchi. Some of these are only globe-like dilatations of the tube itself, and some of the original elements of the tube may be demonstrated in the tissues composing their walls, or even the mucous lining may be more or less intact. Others are undoubtedly cavities originally formed by ulceration, which have become subsequently lined with a false membrane, often very thin, and apparently presenting no traces of the constituents of the tube wall.

With regard to this so-called "false membrane" of cavities, sometimes the lining membrane is quite smooth, about one-twelfth of an inch thick, and apparently a distinct formation, more or less completely independent of the lung-tissue, or the tube out of which the cavity opened. This appearance is strengthened by the circumstance that, without injury to its integrity, one can strip the so-called membrane from the cavity which it lines. But a more critical examination reveals the fact that this impression is erroneous. The free surface of the "membrane," smooth to the naked eye, is found microscopically to be very irregular, and to have projecting from it portions of elastic tissue, which, by examination in another way, can be traced into continuity with the elastic areolæ of the lung. When a section of the "membrane," made vertical to the surface, is examined, it will be seen that there is no distinct membrane, but that the lung-tissue is continuous to the free surface of the cavity, and that the appearance of the membrane is begotten by sharply defined infiltration of the pulmonary tissues surrounding the cavity, almost uniform in thickness, but very thin. Examination of the contents of such a cavity will invariably show the presence of areolæ of elastic tissue, identical with those entering into the constitution of the alveoli; of degenerated pus and lymph cells, of flakes of imperfectly formed fibre material, and of granulo-molecular débris. The structural elements of the fluid contents of a cavity are thus

seen to be identical with those entering into the constitution of its wall, and one can see that the contents of the cavity are produced, not from the true secretions of a real membrane, but from a shedding, more or less continuous, of the infiltrated pulmonary tissues forming its wall.

In summarising the changes in the bronchial tubes as above described, we note that—

1. The cells of the mucous lining are partly shed, sometimes show proliferation, or are wholly wanting.
2. The basement membrane is much thickened.
3. The submucous tissue is generally much augmented in bulk by a fibro-cellular overgrowth, which may form extremely vascular granulation tissue, or well-organised fibroid material.
4. The glands tend, in advanced cases, to atrophy.
5. The cartilages, though late, gradually disappear.
6. There is an enormous increase of fibroid tissue outside the cartilages, forming the major part of the thickened wall.
7. Ulcerations may occur, varying from superficial erosions to deep excavations, or even may convert whole lengths of the tube into irregular, branching cavities.

Alveoli.—We now pass to the consideration of the alveoli. The appearances presented by their walls and contents vary very considerably according as different portions of the lungs are taken for examination.

In many parts, of course, the alveoli presented no disease, in others they were emphysematous or hypertrophied only. It must be here mentioned that a good deal of the lung, which from its size might casually be thought to be emphysematous, is not really so; but there seems to be in many cases a true hypertrophy of the pulmonary tissue, for obvious reasons more commonly seen in the least diseased, or practically healthy lung. Further, it has already been remarked that, the emphysema seemed in some cases to be due to the cutting off of the blood supply, by the obliteration of the vessel leading to the part, although, as far as the emphysema itself went, it differed in no way from the ordinary form. Other alveoli were entirely obliterated, collapsed by the shrinking of the surrounding sclerotic tissue, eventually becoming no longer recognisable. In others, changes might be seen occurring, which at the last resulted in their conversion into fibroid material. These changes will now be described. If a portion of the indurated,

tough, buff or slaty-coloured lung, be taken, and a section made so as to include the edge where it passes gradually, or more or less abruptly, into healthy pulmonary tissue, often the whole process may be traced. Thus the alveolar walls are found to be thickened by fibro-cellular material, partly perhaps originating from the proliferation of their epithelial elements, and partly from exudation from the capillary vessels, which may be seen to be enlarged and tortuous. It is a question whether the fibroid tissue ever really arises by development out of the epithelial cells lining the alveoli, and we think that it occurs rather from the small corpuscular elements, abundantly present beneath the epithelium, in the inter-vascular spaces, and on the walls of the alveoli.

A few of the cells of the epithelial lining were seen shed into the interior, and the alveoli were filled, even in the early stages, with material which was rapidly becoming organised into fibroid tissue. It was unlike the fibrinous exudation of a croupous pneumonia, in being markedly fibroid; again, unlike the cellular stuff of a catarrhal pneumonia, in that there were but very few cells. In fact, it showed definite wavy fibres with a few largish nucleated cells, apparently from the walls, entangled in the meshes. In this stage the walls of the alveoli were still distinctly recognisable, sometimes indeed not much altered, for the thickening described above was not always the first change, but occurred later. What is apparently a further step towards organisation, may often be observed in other parts. Here the fibrous plug may be seen attached to the walls by two or three processes, each containing a small vessel. These tiny capillaries may be seen in many cases perforating the walls, so that the plugs of several neighbouring alveoli are joined together by one vascular network. No doubt these new vessels take their origin from the capillaries in the walls. As one now passes to the completely fibroid lung-tissue, the alveolar walls are traced with greater difficulty, until well-organised fibroid tissue is reached, and no recognisable trace of them is left. These changes can be seen attacking the alveoli of previously healthy lung, or those which had become dilated from emphysema; and in some the exudation, whilst in others the thickening of the walls, was the first change. Generally, however, the two co-existed. At some points the transition from healthy to more or less completely fibroid lung was abrupt, and the gradual changes described above could not be traced. Here probably, for

the time being, the disease was not spreading, but only needed some slight catarrh, causing further exudation, to set going the above process, which would end in a gradual extension of the disease. This distinct change, occurring first in the alveoli, and resulting in the

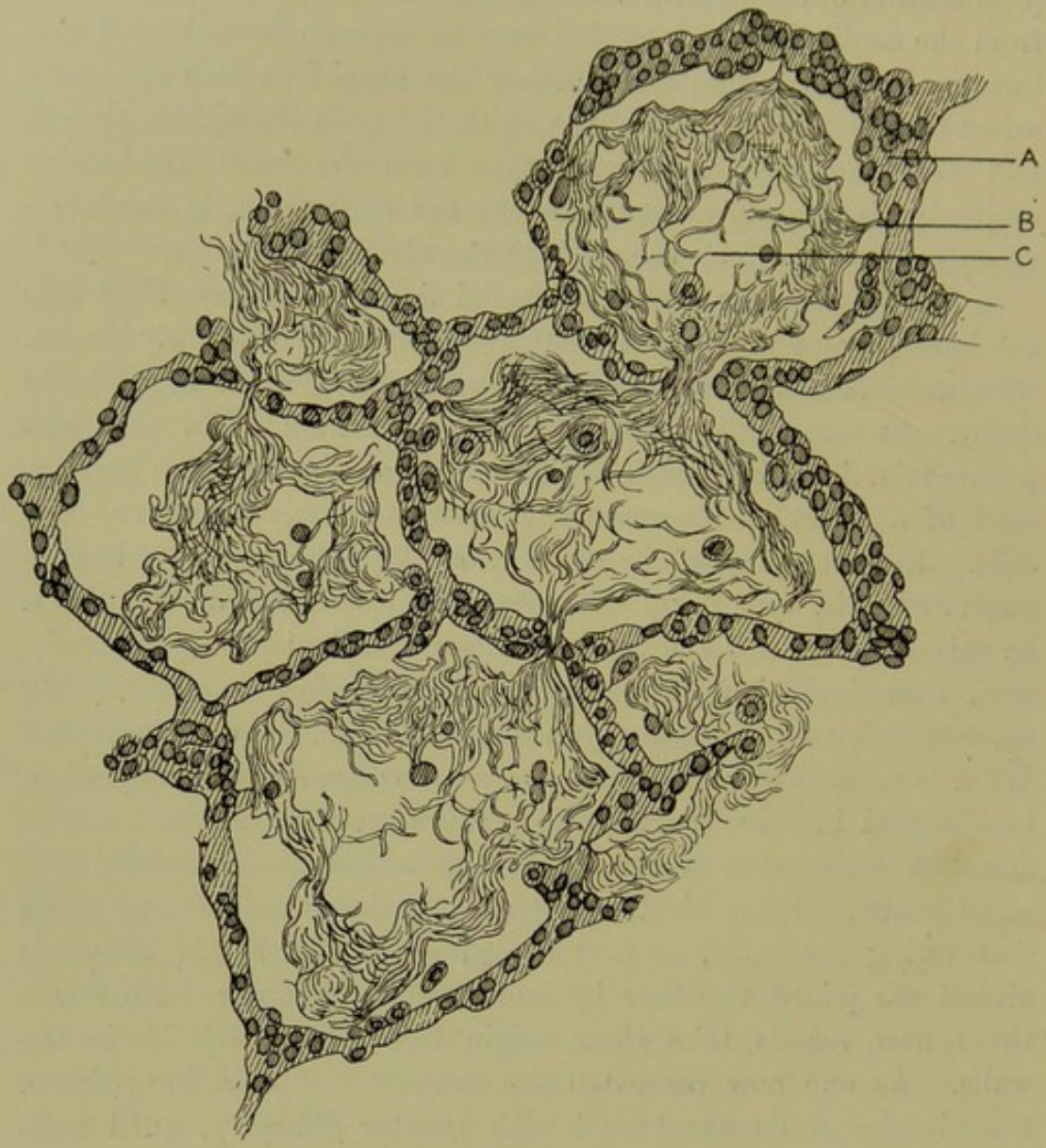


FIG. 2.—Exudation into the alveoli becoming fibroid, showing also slight thickening of the walls; and small vessels from the walls running into the alveolar contents.

A. Thickened alveolar wall.
B. Capillary running from wall into alveolar contents.
C. Plug of exudation becoming fibroid, sparsely cellular.

formation of fibroid lung from their organisation, could not be found in all the cases examined by us. And it must be supposed, that in those cases in which no such change was observed, one of

two things had occurred. First, that the metamorphosis had gone on to such an extent that the origin of it could not be detected, there being at the time of death no fresh areas of disease. Or, secondly, that in some cases the chief change was an interlobular

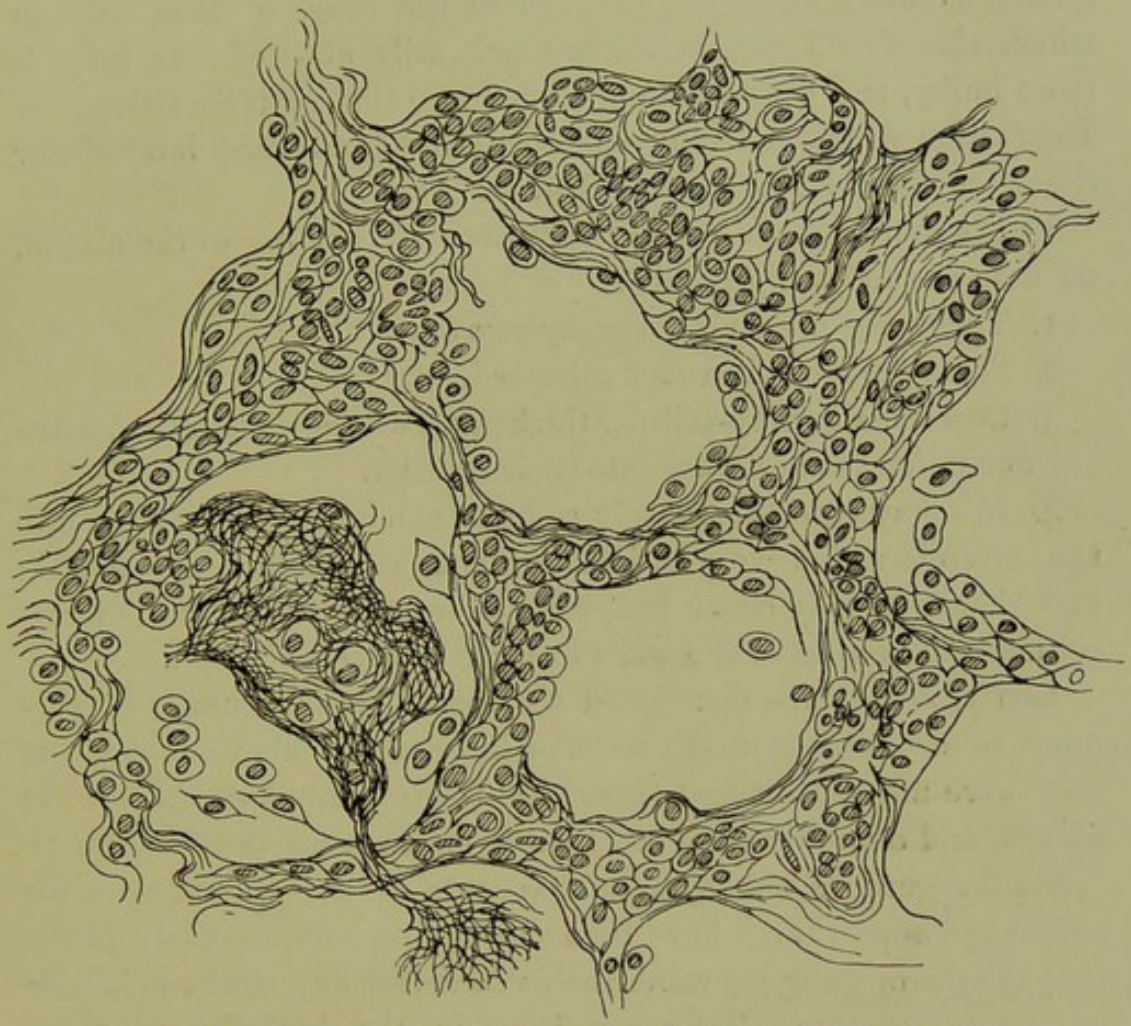


FIG. 3.—Showing marked thickening of the alveolar walls with but little intra-alveolar exudation.

one, in which the alveoli were implicated more by the surrounding contraction, than by disease actually occurring in them; though it was often observed that, in those cases where the interlobular change was the most marked feature of the disease, the walls of the alveoli were considerably thickened by fibro-cellular overgrowth, though there was no exudation within them.* (*Vide* Figs. 2 and 3.)

* It is very difficult to find a proper name for "fibro-cellular" material. Essentially, it consists of fibroid tissue, and of corpuscular particles, more or less abundantly scattered throughout it. According to the hypothetical views of particular observers, these particles may be regarded as nuclear, lymphoid, or

A somewhat similar occurrence may be traced in the cirrhoses of the liver, where in the polylobular variety, large areas are surrounded, and undergo atrophy from the pressure of the surrounding fibroid material, whilst in the monolobular form, each individual lobule is the seat of the fibroid change. It may be observed also that in the lung, as in the liver, in those cases in which the alveoli themselves are primarily affected, the lung is more bulky, and does not tend to undergo that extreme amount of contraction which is often seen where the pleura and interlobular tissue are the chief seat of the disease.

In summarising the morbid appearances occurring in the alveoli, we note the following points:

1. That some are simply emphysematous.
2. Some are shrunken and collapsed.
3. Occasionally fibro-cellular thickening of the walls occurs, without exudation taking place into their interior.
4. In many cases a sparsely cellular exudation takes place into the alveoli, which very rapidly organises into fibroid material; and that this may occur before, together with, or after the fibro-cellular thickening of the walls has taken place.

Lastly, it must be mentioned that occasionally granules of pigment were observed in the walls or interior of the alveoli, but that they were not so abundant there as in the interlobular tissue or the pleura, and certainly were not by any means a marked feature.

With regard to the **bronchial glands** in these cases, after the naked-eye appearances have been noted, it is only necessary to add that microscopically we were unable to detect any marked changes in their structure. They certainly, in the majority of cases, contained a large amount of pigment-granules, but we failed to find any caseation or evidence of softening on the one hand, or undue increase in the amount of fibroid material with hardening, on the other.

Having reviewed the morbid conditions of the various elements of the lung, it will now be necessary to dwell shortly on the cavities occasionally present, and also to consider the appearances of caseous patches or nodules in the fibroid lung.

Cavities.—What has been said in describing the morbid

cellular. Having regard to their whole history, and avoiding every hypothetical assumption, we have agreed to regard them as cellular, and hence the epithet (fibro-cellular) applied to the tissue under consideration.

appearances presented by the bronchial tubes, will serve to show how these sometimes result in excavation of the lung-substance. The formation of cavities in this way, and the structure of their walls, was then discussed, and needs no further description now. There are, however, two other processes which may give rise to this condition. First, some portion of the fibroid material may become gangrenous. This is not of common occurrence, but occasionally, when fresh inflammation supervenes in a lung already the seat of fibroid disease, it does take place. The vessels leading to the part being some of them already obliterated, and the others becoming strangled by the fresh exudation, the portion thus deprived of blood dies, and if the patient should survive, a cavity is formed by its absorption or its discharge in the sputa. The same result may be brought about by the more gradual death of a part. In this case caseation occurs, and by the softening of the caseous material, with its absorption and expectoration, a cavity is formed. In our cases such cavities were of rare occurrence, but the examination of the few small ones which were found showed the following points. In the first place, the vessels leading to the part were obliterated ; and the excavation almost always occurred in dense fibroid material. Much of the caseous material preceding the cavity could be detected around, and also, often remaining in it. In fact, the walls were lined with this cheesy substance, outside which the fibroid material was collected into a more or less distinct capsule. The caseous material was composed of shrunken and epithelial-like cells, free nuclei, nuclear fibre cells, elastic tissue, fat globules, and granulo-molecular débris and pigment.*

Caseous material, either in small patches or distinct nodules, is by no means of constant occurrence, and is never abundant in these lungs. When it is found, it is generally seen in one of two forms. Thus, it may occur as distinct nodules, with softening in the centre—indeed, a cavity is being formed—or, there may be no softening, but a certain amount of calcification has taken place. These nodules are found most commonly in the denser portions of the fibroid material; and whether they are soft or calcareous, it will nearly always be noticed that they are dis-

* The formation and constitution of the false membrane sometimes found lining these cavities have been fully described while speaking of the similar lining of the cavities originating from the bronchi, to which reference may be made here.

tinctly encapsuled by fibroid tissue, so that they have become more or less completely cut off from the rest of the lung. The other form in which caseous material is sometimes observed is more disseminated, having no capsule, occurring in small patches, and most commonly seen in those parts where the fibro-cellular growth is active. Here, to the naked eye, the tissue appears marbled or mottled, and when examined microscopically, amongst the plugged alveoli and increased fibroid elements, small areas may be sometimes observed, consisting of dense masses of lymphoid cells, which towards the centre of the patch have begun to lose their outline, and become granular; or the contents of the alveoli themselves are distinctly caseous, without any patches of crowded lymph cells being observed. With regard to the **presence or absence of tubercular formation**, it may be said at once that the microscope revealed nothing characteristic of tubercle in any of those lungs which have been selected as non-tubercular. It will be remembered that, to the naked eye, no tubercles could be found, and therefore the caseous nodules, in the few cases in which they were present, were the parts presenting the greatest interest in the solution of this question. So much has been said upon this subject, that perhaps it will be of interest to note the steps taken by which we arrive at this conclusion. At the *post-mortem* examination, the fresh caseous material (where found) and scrapings from small cavities, were carefully examined for **tubercle bacilli**, but always with negative results. In one case a cultivation was made of some of this material, but no tubercle bacilli were found. Finally, a guinea-pig was inoculated, but nothing tubercular was afterwards discovered. Next, portions having been hardened in alcohol, sections were made so as to include caseous material and walls of cavities. These were appropriately stained and examined for bacilli, but none were found. Fully recognising the difficulty of finding the bacilli in cut sections, we cut and stained over and over again, but with the same result in all cases; we were, however, able to clearly demonstrate their presence in a fibroid lung which also showed undoubted naked-eye evidence of tubercle. It is also worthy of note that in sections stained for ordinary examination, no characteristic tubercular formation could be recognised. In all the sections of these lungs examined, there were but few large cells, and none to which the title of typical giant cells could be applied. It must not be supposed that the caseous portions

were the only ones examined, but from experience with tubercular lungs, we found that these parts were the most likely to contain the bacilli, and therefore special care was taken to arrive at the truth with regard to their presence or absence there. It has been stated that in some cases of cured tuberculosis of the lung, the bacilli do disappear. Whether this be so or not we are unable to say, but the fact remains that, although the fibroid disease was actively spreading in parts of these lungs, while at others the formed fibroid material was undergoing retrograde degenerative changes, yet in no part could we find tubercle bacilli.

Morbid Anatomy of other Organs.

The Heart.—Some of the important features of the heart have been already insisted on when describing the morbid appearance of the lungs. It remains, however, to complete the account of this organ. Its displacement has been spoken of. In pure fibroid disease the heart is almost always enlarged, and hypertrophy of the walls of the ventricles is clearly observable. It is perhaps more common for the right ventricle to show comparatively more hypertrophy than the left. Some have described areas of fibroid degeneration in the walls of the ventricles, in the valves and columnæ carneæ; these areas to the naked eye appear as greyish streaks and patches, and the knife meets with considerable resistance and toughness in cutting. On microscopic examination there can be seen well-defined connective-tissue elements. The arteries in the neighbourhood of this degeneration sometimes show evidence of thickening and inflammation. The increased size of the heart in fibroid disease is in striking contrast to the diminished size of the organ in cases of tubercular bacillary phthisis, and is one of the principal guides in deciding as to which category the disease belongs. At times one meets with valvular disease, in the shape of thickening of the valves, either due to the general degenerative process, or to some antecedent rheumatic affection.

The Stomach.—When the disease is on the left side, the stomach is universally drawn upwards, and in some cases can be found extending up to the third rib. Fibroid thickening of this viscus has been described, but whether this condition bears any relation to fibroid disease of the lung does not appear.

The Spleen is often enlarged and the capsule thickened. The

organ itself sometimes shows fibroid change, and not uncommonly there is lardaceous disease; but the commonest cause of splenic enlargement in fibroid disease is chronic venous congestion.

The Liver is nearly always enlarged, and when cut into, shows signs of chronic congestion, or fatty infiltration; in some cases there is a well-marked cirrhosis, which presents no difference from the ordinary polylobular variety. It is worthy of remark that, on looking over records, a large number of those cases of cirrhosis of the liver with phthisis seem to belong (so far as the lung condition is concerned) to the tubercular variety of the disease, whereas, one would suppose, that if there be anything in Handfield-Jones' theory of a fibroid diathesis, the lung lesion in these cases would be fibroid, or at all events tuberculo-fibroid; but such does not appear to be the case.

The Kidneys often present signs of fibrosis. They are nearly always congested, and, when examined microscopically, may show evidence of fibroid change. Sometimes they are enlarged; at others, small and granular, the capsule stripping with difficulty. In some instances, again, there may be signs of lardaceous disease.

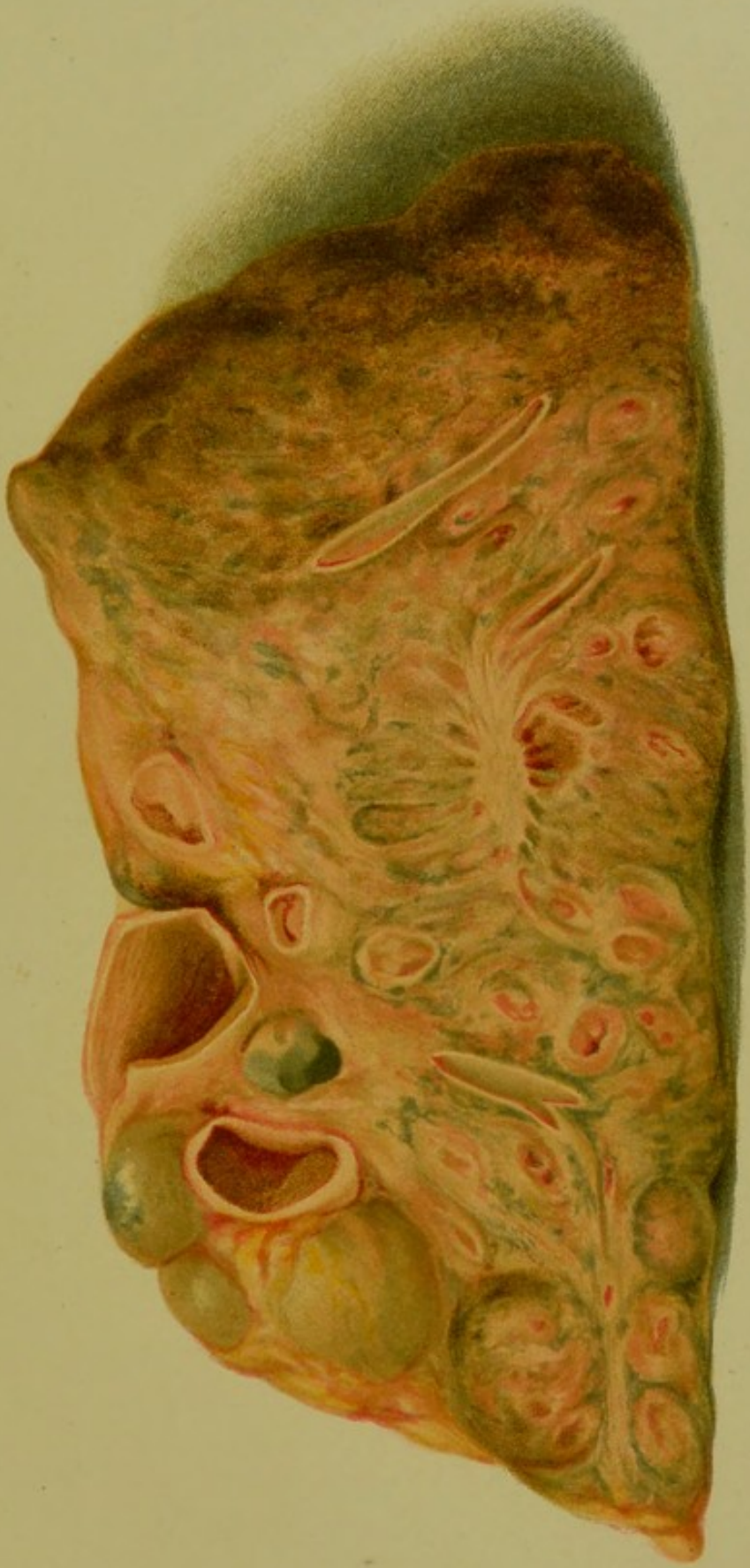
We will now give an account of the *post-mortem* examination of some typical cases.

CASE I.—A. K., aged 2 years, 5 months. (No. 41 in the Table in the sixth chapter.)

This case was one of a child, who having had a rather severe attack of bronchitis when seven months old, with which he was ill seven weeks, had been subject to repeated, less severe, bronchitic trouble, almost continuously since that time. In general health he had at first failed and lost flesh, but subsequently had much improved in these respects, and had so continued until about three weeks before death. Acute bronchitis set in, and he became feverish; the dyspnœa became more and more urgent; cyanosis appeared, and the child eventually died asphyxiated. The physical signs, in addition to those of bronchitis, were almost confined to the right side, there was dulness more or less marked over the whole lung, with cavernous sounds in the upper part. The heart was beating in the sixth intercostal space on the right side, in a line with the nipple. Having thus briefly touched upon the signs and symptoms before death, we will describe the condition as seen on the *post-mortem* table. The autopsy was made about

PLATE III.

Showing a fibroid condition of lung, almost devoid of pigment (some may be seen in the enlarged glands). The bronchi are slightly dilated. The fibroid tissue is not extensive, and is collected in more or less circumscribed masses.



twenty-four hours after death, and rigor mortis was well marked. The body was thin, there was but little subcutaneous fat, yet it could not be said to be wasted. There was no deformity of the chest whatever, the normal shape being maintained on both sides. On opening the thorax, the **left lung** was seen to be much enlarged, and crossed the middle line for about 1½ inches; the hypertrophied edge of the lower lobe overlapped the heart to such an extent that only a portion of the pericardium covering the right side could be seen, about two inches to the right of the sternum. The **right lung** was retracted beyond the costal margin so as to be invisible. There was no excess of fluid in either pleural cavity. A few soft, silky, recent adhesions, easily broken down with the fingers, were found towards the base of the right lung, but there were none on the left side, neither were there any between the pleuræ and pericardium. The condition of the **left lung** may be at once dismissed by saying, that though much enlarged, and hypertrophied rather than emphysematous, it was in other respects quite normal on section; and there was no evidence of tubercle in any part of it.

The **right lung** presented many interesting features. It was contracted to the size and somewhat the shape of a short thick banana, practically airless, and composed almost entirely of thickened and dilated bronchial tubes, bound together by fibrous material, which by its contraction had rendered the intervening lung-tissue collapsed and useless. In this lung hardly a portion could be called normal, and very little that seemed even capable of inflation, or aëration, remained. The tissue was tough, and leathery; it could not be broken down under the finger and thumb; it was buff or greyish-white in colour; it felt heavier than normal lung, but still contained sufficient air, probably in the dilatations of the smaller tubes, to cause it to float in water. Fibrous bands were not marked in this lung, because the whole seemed fibrous. The tubes were uniformly dilated throughout, and towards the periphery were found in some instances to present irregular dilatations. A vertical section, made from the axillary border to the root of the lung, showed the condition extremely well. The smaller dilatations were at the periphery, with occasionally a larger one towards the root. Here, too (at the root), could be seen the bronchial glands, enlarged and firm, but none undergoing caseation, nor presenting distinct fibrous metamorphosis. They seemed out of proportion to the size of the lung, which, of course, was diminished, while they were

enlarged. Around them, and running in some distance amongst the larger tubes, might be seen a considerable quantity of fat, which presented quite a normal appearance. It is also interesting to note that, in spite of the advanced state of the disease, with the exception of the few recent adhesions mentioned above, the pleurá was unaffected, and retained its normal polish, appearance, and thickness. There was no evidence of tubercle anywhere in this lung either. The heart was displaced to the right as a whole, so that the apex remained only half an inch to the left of the middle line, whilst the base extended some two inches to the right of it. The right side of the heart was dilated, and filled with dark clot; the left side was normal. The valves showed no signs of disease. The pericardium was quite healthy. Nothing remarkable was noted in the other organs, but the negative evidence of the absence of any tubercle in any of them is important. Also the absence of any enlargement of the mesenteric glands, and the healthy condition of the intestines must be noted.

Case II.—The next case was that of J. B. (No. 33 in Table in the sixth chapter), who, having presented the signs and symptoms of a contracted and fibroid lung for many years before death, finally succumbed to an attack of acute bronchitis. At the autopsy, the following appearances were noted: The body was well developed, there was no emaciation, nor any œdema. The lips were blue, the veins of the neck were distended, and he was somewhat livid; indeed, he presented the appearances of death from asphyxia, which had been the ultimate cause of his decease, and which was consequent upon the acute bronchitic attack. On opening the body there was no calcification of the cartilages of the ribs. The abdominal cavity presented a normal appearance, there was no peritonitis, and no adhesions; neither was there any excess of fluid.

The right lung was observed to extend one inch to the left of the middle line, so as to cover the heart and completely hide it from view.

On removing the contents of the chest, the left lung was found so firmly adherent over the whole surface that the knife had to be freely used in order to detach it, the pleural cavity of that side being entirely obliterated. The mediastinal tissues were also fibrous and consolidated, and had to be cut from the sternum with the knife. The right lung was likewise somewhat adherent, but to

a much less extent than the left, and there was no fluid in what remained of the pleural cavity on this side.

On more careful examination the various organs presented the following appearances:

Left Lung.—The pleura was considerably thickened, in some parts reaching one-sixth or one-fourth of an inch in thickness. The whole lung felt leathery and the anterior edge solid to the touch, whilst on firm pressure hard, cord-like strands could be felt through the condensed and leathery tissue. The fissures between the lobes were completely obliterated. On making a section into the anterior edge, the fibrous bands which had been felt could now be seen traversing the lung in all directions; and close to the surface in this anterior edge were two small cavities, each about the size of a pea, and filled with cheesy material. Portions of the lung from this part were of a pale (buff) colour, tough, airless, resisting pressure and traction, dry, yielding no fluid on pressure, and sinking in water. Other portions, presenting much the same characteristics as regards consistence, were of a slaty or iron-grey colour.

On cutting into the substance of the lung, fibrous tissue was found greatly increased throughout. The bronchial tubes were everywhere much thickened and considerably dilated. In short, wherever one cut, were seen thickened and dilated tubes (to about the size of a goose's quill) surrounded by fibroid material, and wasted and altered pulmonary tissue. This condition was most marked in the lower lobes. There was no evidence of tubercle anywhere, and the only caseous material found was seen in the two small cavities already mentioned. The bronchial tubes were filled with purulent secretion which freely welled up on pressure. They were dilated uniformly and throughout; but none of them were expanded into globe-like cavities such as may sometimes be seen in these cases.

Right Lung.—Here the chief features were congestion and œdema, the tubes being filled with similar purulent secretion to that seen on the opposite side. But here also the fibrous transformation was commencing, towards the base chiefly; its advance, however, was but slight in comparison with that described on the opposite side. The bronchi showed likewise some slight uniform enlargement and thickening. The free edge, which had been seen extending across the middle line, exhibited marked hypertrophy

and emphysema. There could be found no trace of tubercle in any portion of the lung.

Bronchial Glands.—These were enlarged and juicy, but contained no caseous, or other deposit. Careful examination failed to show any excess of fibroid material in them; indeed, they looked quite normal, though they were unusually large, and perhaps contained rather more pigment than is commonly seen, but were not by any means black.

The Spleen weighed 11 oz., the **Kidneys right** 9½ oz., and **left** 10½ oz., and **Liver** 5 lbs.—They were all enlarged, as will be gathered from their increased weight, and congested, especially so in the case of the kidneys. (There had been albuminuria for some time before death.) But though carefully examined, no other morbid change could be discovered. The capsules of none of these organs were thickened, such as sometimes may be seen in the case of the spleen, and no excessive firmness or fibrous induration could be made out. It must also be observed that they were not lardaceous.

The Stomach and Intestines presented no abnormal appearances, neither could any changes be detected in the mesenteric glands, which appeared quite healthy.

The Heart.—This, as far as its walls and valves were concerned, was perfectly healthy. The death from asphyxia had given rise to great congestion of the right side, which was full of dark blood and clots. The pericardium, however, was markedly affected, being firmly adherent, on the one hand, to the surface of the heart, the cavity being entirely obliterated; and, on the other, to the left lung; and was matted by fibrous material with the other mediastinal tissues.

Lastly, it is important to note the absence of any appearances which, as far as we could determine, showed any sign of tubercular formation throughout the entire body.

Case III. (No. 12 in the Table in the sixth chapter) is that of a young man aged 24 years, who had been suffering from fibroid disease for fifteen years. He had been repeatedly under observation for the last four years, and during that time he was always in good health and able to follow his occupation of ship steward. On his return home from his last voyage, in May 1893, he was suddenly attacked with hæmoptysis, losing blood to the extent of two pints. He recovered from this, but on the following day he

had another attack as severe as the first, and died suddenly in the act of bringing up the blood. His was a case of right-sided disease; the heart was beating one inch outside the right nipple line, and in the fifth interspace. There was considerable contraction of the right side of the chest, and evidence, over the whole of that side, of fibroid disease. The necropsy was made twenty-four hours after death. There was a good covering of muscle and fat over the chest. The cartilaginous portions of the ribs were not calcified, except those belonging to the first and second. On removing the sternum, the following appearances were found:— The left lung was enormously increased in size, and extended fully five inches to the right of the sternum, its anterior edge dipping down into the right cavity of the chest. By this arrangement the anterior mediastinum was entirely hidden from view. But on lifting up this portion of the left lung there was no middle mediastinum beneath it, the only structures between it and the spine being the trachea, aorta and œsophagus, and a small part of the mediastinal tissues. The heart, pericardium, and greater part of the mediastinal tissues were drawn over and fixed in the right cavity of the chest. The heart was found in a situation corresponding to the position made out during life through the chest wall. The apex lay an inch outside the *right* nipple line, and in the fifth interspace; the base of the heart reached up to the fourth rib, and was situated about an inch and a half away from the right edge of the sternum. The pericardium was firmly bound down to the right lung, and on attempting to pass the hand into the right pleural cavity, it was found to be entirely obliterated, and occupied by firm, tough, and old fibrous adhesions. The right lung could be felt lying at the back of the cavity, close to the spine; it was small and hard to the touch, and great difficulty was experienced in extracting it. The adhesions were so firm and dense, that the organ had to be cut out of the chest. There were no adhesions worth speaking of in the left pleural cavity. The heart and lungs were removed together, and on closer examination the following morbid changes were observed:

The Heart and Pericardium.—As has been said, the external surface of the pericardium was firmly bound to the pleural surface of the right lung, uniting the two in one mass. The pericardium was thicker than normal; on opening up the sac there was but a slight excess of fluid noticed, and no adhesion or white spots, the

visceral surface of the membrane presenting a smooth and shining appearance. The heart itself was enlarged, but the increase in size did not seem to be more on one side than the other. On opening its chambers, there was a marked absence of blood. The wall of the right ventricle was a little thicker than normal, and the left ventricle was hypertrophied, perhaps, to a greater extent. The cardiac muscle looked pale, but no spots of fibroid material could be detected. The valves were all of them competent and healthy, and the aorta contained no more evidence of atheroma than is generally found in adult life. The heart contained no excess of fat. The mediastinal tissues were matted together, and the glands and other structures composing them, were indurated.

The left lung was voluminous, and did not collapse to any extent when the pleural cavity was opened. It was fully twice the normal size. The pleural covering was not thickened, but the interlobar fissures were obliterated by old and firm adhesions. The colour of the lung did not depart from what is generally observed in people who have passed much of their life in towns; it had a dull bluish look, with darker lines of carbon pigment. The edges of the viscus were rounded, and on pressure being made with the finger, deep pits were produced. At the base there were four or five large bullæ about the size of a pigeon's egg, and smaller ones were found at the anterior edges and apex. On palpation the organ crepitated under the finger, and here and there, scattered about, could be felt hard masses in the lung-substance. When cut into, a considerable amount of blood-stained serum escaped from the base. There was great congestion at the inferior parts, and here and there, small areas of blood-stained pulmonary tissue simulating infarction. The emphysema over the whole lung was manifest. The bronchi were not dilated, but they were slightly thickened, and the mucous membrane was congested. The vessels appeared dilated, but no marked thickening of their coats could be observed. There was no overgrowth of fibrous tissue in this lung, at least so far as could be determined by naked-eye inspection.

The hard masses in the lung-substance on section, appeared to be patches of caseous material, masses of fibroid tissue, and cavities containing putty-like matter. In the anterior edge there was a caseous centre as large as a bean. At the apex there was one

small encapsuled patch as large as a pea, and in the apex of the lower lobe there was found a large, irregular, smooth-walled cavity, filled with blood. This cavity led into a bronchus, but its walls were not like those of a bronchus. These were the principal morbid changes to be seen in the left lung.

The right lung presented a very different appearance. The pleura was one-third of an inch in thickness, and at the apex and base very much thicker; in these situations it was fully an inch deep, and the lung seemed to have been encroached upon by its covering. In the pleura were some large irregular spaces, with strands of lymph running across them; one especially large space ran down from the apex to within an inch or two of the base. The lung itself was about the size of the closed fist, and was composed mainly of dilated tubes and fibroid tissue, there being very little lung-substance proper left. All trace of division into lobes was gone, and the lung had a compact elongated shape, resembling, in external conformation, the spleen. The fibroid tissue was collected in large quantity at the root of the lung, and the main bronchi and vessels were encased by it. Thence it spread out and invaded the lung in all directions. It was particularly plentiful around the bronchi and vessels. Some of the fibrous tissue also appeared to spring from the pleura, and could be seen as thick white lines dipping down into the interlobular spaces. There was very little pigment to be seen. The lung, to the touch, was intensely hard, and when cut into gave almost a sensation of cartilage. It was difficult to say which part of the lung presented the most complete change, for the organ as a whole was affected, and the disease had advanced to such a degree that one part of the lung could not be pointed out as being more affected than the other.

The bronchi were everywhere uniformly dilated, the walls of the tubes were much thickened, and the mucous membrane showed signs of chronic inflammation.

The right bronchus, soon after entering the lung, presented a large oval-shaped dilatation, from which were given off the bronchial tubes to the lobes in the lung. The smaller bronchi could be followed with but little difficulty to their terminations, near the pleural surface of the lung. From the size of the organ and the number of dilated tubes, it would seem that many of them had become obliterated, and on carefully inspecting the walls of the tubes, minute holes could be distinguished, just admitting

of a probe, and which seemed to end blindly. The tubes were filled with froth and blood-stained phlegm.

The vessels of this lung, so many of them as remained, were thickened, but not dilated. The right pulmonary artery was a little narrowed, and its wall was certainly thicker than the left. On following it out to its entrance into the lung, however, the vessels proceeding from it were immediately narrowed, and in many cases obliterated, looking very much as if they had been ligatured.

A marked feature of this right lung was the almost complete absence of cavities, other than bronchial dilatations and areas of caseous deposit. On looking carefully through the lung, one or two spots of caseation could be found; they were situated in the centre of fibroid masses, but they were not numerous enough or large enough to account for the whole of the fibrous transformation.

Lastly, to complete the description of the lung, the **bronchial glands** must be mentioned. They were enlarged, hard and firm, and enveloped in thick, tough capsules. There were no areas of softening to be seen, but an excess of fibrous tissue was quite evident to the naked eye.

All that could be said of the other organs was, that the kidneys appeared somewhat enlarged and congested, and that the same remark applied to the liver and spleen.

Case IV. (No. 45 in the Table in the sixth chapter), although by no means typical of pure fibroid disease of the lung, yet illustrates many of the points raised in the chapter. The lungs were taken from a woman, aged 44, who had complained of winter cough for nearly twenty years. During life there was flattening at both apices, with dulness and bronchial breathing; but no displacement of viscera. When the necropsy was made, the pleura was slightly thickened over both apices, and much more so at the bases, the lobes were also bound down by adhesions. This pleural thickening was much greater over the left lung. The lungs, to the touch, were crepitant at the bases, and only slightly so at the apices. In this latter situation lumps about the size of a filbert could be felt.

Right Lung.—This lung on section was like leather, and it could at once be seen that the apex was the chief seat of the disease. Its colour was dark, and numerous broad, white, intersecting lines of fibrous tissue running in all directions were plainly visible. The bronchi stood out prominently, their walls were

thickened, and surrounded with aggregations of fibrous tissue. The absence of marked dilatation of the tubes was a special feature of this case. In no instance could they be said to be more than slightly dilated; indeed, thickness of the walls of the tubes was much more in evidence than bronchiectasis. The vessels were dilated, but not much thickened.

Beyond the numerous white lines of fibrous tissue mentioned above, there was not much to be found anywhere, and the base of the lung was wholly free from it. With the exception of the apex, there was no diminution in the size of the lung to be noticed. The pigmentation, such as there was, was chiefly confined to the apex.

There were several caseous deposits found in the apex of the lung, but no cavities. The deposits were as a rule circumscribed, and surrounded with fibrous tissue. The caseous matter was submitted to tests for tubercle bacilli, but without any positive result.

The left lung does not need further description, for in all essential particulars it was similar to the right.

The heart was not displaced, there was no hypertrophy. The muscular wall was thin and pale, but there were no spots of degeneration in it. The valves were healthy, and the pericardium was not adherent, either to the heart or the lung. The pulmonary artery seemed to be unduly dilated, and when its branches were followed into the lung, the same appeared to be the case there. No thickening could be discovered.

The bronchial glands were much enlarged; they were of intense hardness, and had thick well-defined fibrous capsules. On cutting into them they were black in colour, and there was evidence of radiating bands of fibrous tissue in them. In many parts they were calcareous.

The kidneys showed evidence of interstitial change, and the liver was congested and cirrhotic.

CHAPTER IV.

CLINICAL ACCOUNT OF FIBROID DISEASE UNACCOMPANIED BY TUBERCLE.

In treating of this important division of the subject, it will be most advantageous to adhere to the plan which has been followed in the chapter on the pathology of the affection; that is, to give first of all a description of the disease as it presents itself in its clinical aspects, and then to relate some of the most typical cases which have come under our observation, insisting strongly upon the precise points which enabled us to form a definite opinion as to the true nature of the disease. This method of drawing inferences from both generals and particulars, is of peculiar value when the clinical study of a disease is in question; for, from the nature of things, there must be a marked deficiency of diagnostic points in an actual case seen at the bedside, when compared with the multitudes of signs and symptoms finding an appropriate place in a general account, including all variations of the disease.

The account in this chapter will aim at being clinical in its broadest sense, embracing everything that can be elucidated concerning the disease: its origin, signs, symptoms, diagnosis, prognosis, complications, and sequelæ; the family history, the ages of the patients;—all these separate headings will find a place in this clinical account of pure fibroid disease of the lung. The consideration of the clinical side of fibroid disease is of the deepest interest, for it is when approaching it from this standpoint that misconceptions as to its real nature most frequently arise. We cannot help thinking that, if the special diagnostic points of fibroid disease were better understood and borne in mind, less confusion would occur, and much would be done towards the general acceptance of this disease as a clinical entity, apart from tuberculosis.

From the point of view of the patient, a just appreciation of this part of the subject is all-important; for the fibrotic has a much

longer prospect of life, and a much better prospect of working during that life, than the tuberculous patient. He seldom requires the changes abroad, or the special care at home, which are so necessary for the well-being of the phthisic.

Remembering this, let us now pass to a description of the ætiology, signs, symptoms, diagnosis and prognosis of the disease, taking them seriatim.

Ætiology.—On looking over all the assignable causes of this affection, we have a lengthy catalogue of conditions, all of which may be regarded as capable of giving origin to fibroid disease of the lung. Some are a fruitful and common source of the disease, whilst others belong to the domain of single recorded instances, found scattered over the literature of the subject. The various causes may be thus tabulated:

1. Broncho-pneumonia following upon whooping-cough or measles.
2. Acute croupous pneumonia.
3. Prolonged chronic bronchitis.
4. Pleurisy.
5. Inhalation of irritating dust.
6. Collapse of the lung.
7. Syphilis.
8. Bronchiectasis.
9. Traumatic causes.
10. General fibroid state (fibroid diathesis).
11. Alcohol.

Each of these causes requires separate consideration.

Broncho-pneumonia.—By common consent the first of these headings—viz., broncho-pneumonia succeeding upon whooping-cough or measles—is regarded as a frequent mode of onset of fibroid disease; indeed, there are few writers on this subject, who do not mention it as accounting for a large proportion of the cases met with. And, so far as our observations of young adults affected with the disease are of value, we should entirely endorse the view of the majority of writers. But the huge preponderance of this cause over all others, is only met with in those cases which have begun in early life. It finds no such prominent place in those which have come on comparatively late in life; and this is no more than one would expect. Broncho-pneumonia dependent upon measles and whooping-cough being excessively rare in adult life, indeed, almost unheard of, it would be strange if any cases of fibroid disease, arising at that period, were attributable to that cause. So that, for

those cases arising later in life, some other cause must be sought. But another fact of great importance in connection with this subject is, that few cases, having begun in early life, and having lasted well into the middle decades, are due to broncho-pneumonia. From this it may be inferred that few of those so affected in the earlier periods of life live to a great age.

How often this broncho-pneumonia, following whooping-cough or measles, is answerable for fibroid disease, will be readily seen by referring to the Tables of the cases coming under observation, in the sixth chapter. Here, with a frequency which is surprising, and quite unaccounted for by chance circumstances, broncho-pneumonia following whooping-cough or measles, is over and over again assigned as the beginning of the disease. The records of Children's Hospitals also show the potency of this cause; though it must be remembered that by far the greater number of the cases quoted in the Tables are those beginning in childhood.

It would be profitable to inquire the after-history of children attacked with broncho-pneumonia following whooping-cough or measles; to know what proportion become affected with fibroid disease, and how many succumb to tuberculosis; but, so far as we are aware, there are no data on which conclusions of any value could be based.

The history given by patients who owe their disease to this cause is quite definite, and is somewhat as follows. After an attack of whooping-cough or measles, instead of completely recovering, they become rather suddenly affected with a new set of symptoms, in which cough, dyspnœa, and fever play a prominent part. On examination of the chest at this period, dulness and bronchial breathing, attended with crepitations, and sibilant rhonchi, can generally be found over various parts of the lungs. The child, after suffering for a variable time with this attack of broncho-pneumonia, at length recovers to a certain extent; but if fibroid disease of the affected lung be about to ensue, the injured organ fails to heal, and the recovery is never perfect. Although the child may regain a fair amount of health and strength, yet the cough, dyspnœa, and expectoration never altogether cease, and after a time, evidence of the accession of fibroid disease becomes manifest. It is held by Eustace Smith and others that, in an attack of broncho-pneumonia, the bronchial tubes in the affected part of the lung quickly lose their tone and become

dilated, never regaining their calibre; and Wilson Fox suggests that this may be the starting-point of the fibroid process. But more light is needed upon this subject. What determines the onset of fibroid disease after broncho-pneumonia, in some cases and not in others, cannot with any accuracy be accounted for, unless we assume a diathesis.

Acute Croupous Pneumonia.—Concerning the frequency of acute croupous pneumonia as a cause of fibroid disease, there has always been expressed much contrary opinion and much doubt. Some have denied altogether its place in the ætiology of fibrosis of the lung, while others have assigned to it more importance than it really deserved. There are, however, on record many cases in which the clearest sequence of events is traceable from an attack of acute pneumonia, to the establishment of a fibroid process in the lungs. We must again refer the reader to the Table of Cases in the sixth chapter; he will there find that a not altogether insignificant minority give evidence of being due to such an attack. Much of the scepticism concerning acute pneumonia as a cause, is doubtless due to the well-known remark of Chomel, who only met with it eight times in a pathological experience extending over thirty years. In most instances, the patient was in perfect health until he had an attack of acute pneumonia; this, instead of resolving in due course, went on smouldering, and the lung began to show signs of fibroid disease, affecting chiefly the part which had been attacked by the pneumonia. It may be said that, although by far the majority of cases of acute pneumonia do not terminate in fibroid disease, yet enough of them do so in order to make it, next to broncho-pneumonia and bronchitis, the most frequent exciting cause of the process.

Prolonged Chronic Bronchitis is also recognised as an important factor in the causation of fibroid disease, but whereas we saw that broncho-pneumonia was answerable for most of those cases occurring in early life, bronchitis is chiefly effectual in inducing fibroid disease coming on late in life, and at this period is responsible for almost as many cases as broncho-pneumonia was in the earlier ages. For bronchitis to be able to produce fibroid disease, it must be prolonged; a few years seem to make but little impression upon the lungs—indeed, most individuals, attributing the beginning of their disease to this cause, give a history of fifteen or twenty years. or even longer; and if this be

so, it would be impossible for cases coming on early in life to be caused in this way. We may stop to inquire the reason for the sequence of fibroid disease upon prolonged chronic bronchitis. Of the various views that have been offered, the most probable seems to be that upheld by Iuergensen, in his article on Interstitial Pneumonia in Ziemssen's *Cyclopædia*, for which explanation the reader is referred to the historical chapter. He there maintains that the secretion of the tubes, caused by inhalation of dust, sets up inflammatory change, and that in lungs affected in this manner, the large quantities of foreign material hinder the flow of lymph through its channels; and the secretion, which before did not remain a sufficient length of time to cause inflammatory change, now has ample opportunity to act injuriously on the lung-tissue. This is also true of cases where dust does not play such an important *rôle*. It is also held by Iuergensen and others that the mere chronicity of bronchitis is enough to cause induration of the lung. We may, therefore, suppose that either the induration is brought about by secretion of the bronchial tubes acting upon a lung whose nutrition is impaired by impeded lymphatic flow, or by the mere chronicity of the bronchitis.

Pleurisy.—Coming now to pleurisy as a factor in the ætiology, we must consider its agency in the production of fibroid disease in two different lights: first, as a dry pleurisy; second, as a pleurisy with effusion of either serum or pus. The first of these two conditions must, we think, be answerable for almost all the cases of fibroid disease supervening upon this affection; for, as we shall presently show, there is no reason to believe pleural effusion effective in bringing about such a result. Though a dry pleurisy is sometimes undoubtedly a cause of fibrosis, yet we cannot help thinking that its frequency, ætiologically, has been somewhat overestimated. Because, at a *post-mortem* examination of fibroid disease, the pleura is found enormously thickened, that is hardly a proof of the disease having begun as pleurisy. And, again, it must be remembered that two of the common causes of fibroid disease—viz., broncho-pneumonia and acute croupous pneumonia—are almost always attended with a deposition of lymph on the pleura. Yet an attack of dry pleurisy, altogether independent of lung complications, does sometimes pass into fibroid disease. This sequence of events has been noted several times by one of us. A patient comes complaining of a dull, heavy aching in his side; on examination,

he is found to have a dry pleurisy. This does not get well, and after a time there is evidence of fibroid disease. This is apparently the history of not a few cases of fibrosis of the lung. With pleurisy with effusion the case, however, is very different. We cannot recall a single instance of fibroid disease which gave a history of ever having had a pleural effusion; and on calling to mind those cases of effusion which have come under our notice, we cannot refer to a single one as having passed into a fibroid condition. Again, the disease rarely follows upon empyema. Inquiry into the after-histories of those who have had pus in the pleural cavity, yields negative results, so far as fibroid disease of the lung is concerned. After empyema, one often finds contraction of the chest and diminution of the respiratory murmur, but no evidence of fibroid disease. Dr. E. B. Hastings, the late resident medical officer at the East London Hospital for Children, has taken great pains to obtain the subsequent histories of all the cases treated for empyema at the hospital; among these records we do not see a single case of fibroid disease, and no stronger proof of its rarity as a result of empyema could be given. So that, while dry pleurisy may be a cause of fibrosis, there is not much evidence to show that, effusion of serum or pus in the pleural cavity, is followed by it with any frequency.

Inhalation of Dust.—With regard to the inhalation of irritating kinds of dust, not much need be said; most are agreed as to this being a cause of fibroid processes in the lungs. In those regions where people are much exposed to these irritating dusts, the lungs, after an antecedent stage of bronchitis, often take on fibroid induration which presents no difference from the ordinary forms. The probable mode of operation of the dust, producing this effect, we have already discussed, when speaking of ordinary chronic bronchitis. We, who draw our observations from London and its surroundings, have but little opportunity for studying this element in the production of the disease, since here it is unfrequent; but in towns where the occupations of workmen render them peculiarly liable to inhale dust, cases of fibroid disease from such a cause are by no means uncommon.

Collapse of the lung, independent of pleural effusion, we have mentioned as a cause, on account of our having met with one case which could hardly be regarded as having begun in any other way. So far as we know, Dr. Wilson Fox is the only other observer

who discusses the probability of this condition inducing the disease. It is quite conceivable that after a portion of the lung has become collapsed permanently, a growth of fibrous tissue might spring up in the injured portion, and become the starting-point for a widespread fibrosis of the whole lung; and apparently in some few instances this is really what does happen.*

In the case above referred to, which will be found in the Tables in the sixth chapter, a child, who was in perfect health, went out to play. Suddenly, without any warning, a violent fit of coughing came on, the child became blue in the face and gasped for breath; from these violent symptoms she recovered, but a persistent cough with much expectoration always remained, and at the time of observation there was well-marked fibroid disease of the lung. We may even go a step further, and regard the partial fibroid state of lungs which have long been pressed upon by chronic pleural effusions, by tumours and aneurysms implicating the bronchi, and by plugs of mucus in the bronchi in chronic bronchitis, as due to the deposition of fibroid material in the collapsed portions, aided by the chronic congestion of the parts; though a perfect condition of fibroid disease is never produced by these means. So also this quasi-fibroid state may be induced in a collapsed portion of the lung brought on by the pressure of an enlarged auricle on the bronchus or lung, in heart disease.

Syphilis is undoubtedly, ætiologically, concerned in fibroid disease; but it is with extreme rarity that cases capable of being ascribed to this cause are to be met with. Cases of syphilitic disease of the lung are not wanting, but hardly any of them could be looked upon as perfect examples of pure fibroid disease of the lung; most of them taking the form of patches of gummatous material with induration around them. In some few cases a diffused form is met with, much resembling pure fibroid disease.

Bronchiectasis.—Coming now to dilatation of the tubes, it is questionable if it should find a place among the diseases which give rise directly or indirectly to fibroid disease of the lungs. It has elsewhere in this work been stated that bronchiectasis is a result, not a cause, of fibroid disease. Prior to Corrigan, the generally accepted view was that, the bronchiectasis was the cause of the disease, and at the present time there are not wanting those who

* In this way, it is possible that fibroid disease may occur as an indirect result of pleural effusion; but of this we have met no instance.

still hold to the belief in the possibility of such being the case, in some instances at least. Biermer and Grainger Stewart may be mentioned as regarding this as possible. Dr. Bastian has ably dealt with this question, and has offered weighty reasons for believing bronchiectasis to be a result and not a cause of the lung condition. Still the fact can escape no one who has paid particular attention to fibroid disease and allied conditions; that in those cases in which the bronchiectasis is the prime factor, the intervening lung-tissue presents evidence of a certain amount of fibroid deposition; scanty, it is true, but nevertheless quite evident. In these cases it is difficult to escape the conclusion that this fibroid change arose around the bronchi as the result of their abnormal size, and consequent surrounding chronic inflammation. Not long ago, we performed an autopsy upon a case of this kind, in which the whole of the lower lobe of the right lung was traversed with bronchial tubes the size of the little finger, and the intervening lung-tissue was in an incipient state of fibrosis. It will be remembered what was said about dilatation of the tubes in broncho-pneumonia, and the possible *rôle* played by them in the production of fibroid disease. But, with these exceptions, it must be admitted that in the majority of cases in which there is bronchiectasis, it is due entirely to the fibroid disease.

Traumatic Causes.—Occasionally cases are met with in which fibroid disease is clearly traceable to an injury received in the chest, the ribs being broken, and the exposed ends entering the lung and causing laceration. When such cases come to the *post-mortem* table, the pleura is found strongly adherent at the place where the injury occurred, and a portion of the rib may be found imbedded in the cicatricial mass. The fibroid process can be seen extending from the point of injury into the lung, and invading it in all directions. One such case may be here briefly referred to, though unfortunately the notes taken at the *post-mortem* examination are missing. A young man, aged 23, of unusually fine physique, and in excellent health, was squeezed between the shaft of a cart and a wall, the shaft pressing upon the fifth and sixth ribs on the left side. It could not be ascertained that any ribs were broken, but soon after the injury he began to cough and expectorate. Emaciation to a slight extent set in. On examination, one or two years afterwards, the whole of the left chest was much contracted, immobile, and dull on percussion. Auscultation showed bronchial breathing at the lower

parts of the lung, and large, coarse crepitations. The young man remained in much the same condition for a year longer, and then quite suddenly developed hectic and rapid emaciation. A pneumothorax of the left side was noted, and he died a short time after. At the *post-mortem*, the pleura was enormously thickened at the level of the sixth rib in the axilla, and in this mass of fibrous tissue was found a piece of exfoliated rib, penetrating the lung. From this centre, fibrous tissue spread out in all directions throughout the lung. The bronchi were not much dilated. There were no tubercles in this lung. The opening into the pleura causing the pneumothorax could not be discovered. The right lung, from apex to base, was stuffed with fresh miliary granulations.

Another instance of fibroid disease originating in traumatism was the case of Daniel Gilbert, a picture of whose lung is reproduced in this work, and which is an excellent example of this mode of origin.

There can be no doubt that the fibroid process in these cases was set going by the injury. The state of the parts revealed at the *post-mortem* all supported such a supposition. Yet it is a very rare occurrence, and only a few cases are on record.

Fibroid Diathesis.—We come now to discuss the question of a diathetic condition, favouring the onset of fibroid disease. It may well be asked, Why is it that some cases, after various attacks of diseases of the lungs, degenerate into fibroid phthisis, while many others escape without any untoward result? The question is hardly capable of answer, unless it be assumed that there is a fibroid diathesis which renders some prone to develop fibroid disease, while those who do not possess this vulnerability, escape. We have seen in the historical account that Handfield-Jones believed strongly in this diathesis, and went even a step onward and looked upon the lung fibrosis as merely a local manifestation of a general disease. As is well known, the arteries, the heart, the liver, and the kidneys, in addition to the lungs, are liable under suitable conditions to become fibroid. May we not suppose that these individual tissue metamorphoses are governed by a general fibroid diathesis, which comes into operation when one or other of the organs has its vitality lowered, or its nutrition in some way altered, so as to allow of the prevailing tendency to take effect? Or are these fibroid changes in different organs quite independent of a diathesis, and do they rest for their production simply upon

lowered vitality, or altered nutrition of the organ? If the first of these suppositions be accepted, one would expect to find sometimes several sets of systems affected at the same time in the same individual; and Handfield-Jones seems to have shown this to be the case. In this way could be explained the high tension pulse, the hypertrophied heart, and the fibroid transformation in the liver, spleen and kidney, sometimes met with in fibroid lung disease. On the whole, this seems more capable of credence, than to suppose that the result was brought about simply by an alteration in the metabolic activity of the organ, without the intervention of a diathesis.* It is by no means improbable, however, that a fibroid condition of one system might favour a like event in another. For instance, a patient with arterial fibrosis would be, on that account, in a position to turn the scale in favour of the onset of fibrosis in the lung, kidney, or liver, when these organs had their nutrition interfered with, or *vice versâ*. Whatever view be taken of this difficult problem, we must remember that, all those lung conditions, which have been described above as being to a certain extent responsible for fibrosis, can only be viewed in the light of direct exciting causes; as it were the lighted match which produces the explosion. When we come to speak of the family and personal history of these cases, we shall inquire what evidence there is of a diathetic fibroid taint.

Alcohol.—In connection with ætiology there yet remains to consider the influence of alcohol. Sutton inclined to the view that the abuse of alcohol was capable of exerting a direct effect on the production of fibroid disease. Several facts favour such a view. In the first place, in an analogous condition, the well-known effects of gin-drinking on the liver and kidneys may be instanced; secondly, some cases have come to hand in which tubercular phthisis has apparently taken on a fibroid change through the agency of imbibing large quantities of alcohol; and, lastly, it has been stated by some, that croupous pneumonia is less likely to resolve, and more likely to pass into a fibroid state, if the therapeutics have consisted in a liberal use of alcoholic stimulants.

* On the other hand, one of us thinks that the increased work to which the heart is subjected, is sufficient to account for its hypertrophy, more especially, as has been noted, that hypertrophy is relatively greater on the right side. Further, he thinks that the changes in other organs can be well explained by the chronic congestion to which they are subjected, on account of the back pressure.

That alcohol is an excitant may certainly be true in some cases, but it must be noted that fibroid disease of the lung often comes on at a tender age, at a time when there could be no possibility of alcohol playing any part in the production of the disease. For these reasons, alcohol must not be regarded as a general and direct cause, but only as a special and indirect one, operating upon, and giving the *coup-de-grâce* to a constitution already predisposed to fibroid disease.

In closing this account of the ætiology of pure fibroid disease of the lung, it will not be out of place to summarise the different points which are claimed to have been brought out by the inquiry. The statistical part of this subject will be seen on reading the tables and analyses to be found in the sixth chapter. We may then, recapitulate our results as follows :

1. There is reason to believe that pure fibroid disease of the lung depends, in common with like affections of other organs, upon a vulnerability, or susceptibility of the organism to take on a fibroid process, when the particular organ to become so affected has, by some means or other, its vitality lowered and its nutrition altered. This vulnerability is termed the "Fibroid Diathesis."

2. The causes which operate in producing this lowering of vitality, and alteration of nutrition, are chiefly those diseases which we have discussed and enumerated.

3. Of these diseases, the most common are, broncho-pneumonia, acute pneumonia, and bronchitis.

4. The abuse of alcohol may be said to exert some influence over the production of the fibroid state.

5. Some of these diseases set going the process in early life, while others are responsible for its onset in the later periods. Thus, those which give rise to fibroid disease in early life are, broncho-pneumonia, acute croupous pneumonia, collapse of the lung, and pleurisy. Those giving rise to it later in life are, bronchitis, bronchiectasis, traumatic causes, alcoholic abuse, syphilis, and sometimes acute pneumonia and pleurisy ; the two last acting either in early or late life.

Family History.—Let us now pass on to consider the family history of people affected with fibroid disease, with a view to determine if there be any family taint, or condition, which specially influences it. First, with regard to the question of a family history of tubercle. It will be seen in the chapter on the

analysis, that 13 out of 45 cases gave a history of tubercle in some or other members of their family. But what is a family history of tubercle worth? To arrive at a right appreciation of the extent of tubercular antecedents in a family, is well-nigh impossible. The knowledge possessed by patients on this point is meagre and inaccurate to the last degree. It is difficult to meet with an individual who can give even a correct account of the cause of death of his dead relatives, in his own generation, to say nothing of the generation which preceded him. Tubercle presents itself in such a multitude of different ways, that any one not versed in medicine, is very likely to forget some of its manifestations. Again, wasting and cough are taken by the laity to represent a consumption, or, colloquially, a "decline." Further, there is often on the part of patients a marked reticence with regard to the admission of a history of tuberculosis, many hesitating to brand themselves, so to speak, with a taint, which might lead the medical adviser to take a more unfavourable view of the case. In connection with the subject of heredity, one may recall to the reader's mind the passage in Blackstone's *Commentaries*, where he shows that, by the time one has gone back to the twentieth generation, a man must have had above a million ancestors. These facts, taken together, will show how much value can be attached to a family history of tubercle, and 13 out of 45 cases giving such a history would be by no means excessive, if they were all normal, healthy individuals.* So, from the collected cases we cannot regard it as proved, that there is an undue amount of tubercular taint in the family histories. And the recorded observations of other workers in the same field give a like result.

It must be understood, however, that we do not deny the influence of heredity in tuberculosis; we are simply endeavouring to show that, when looked at with this end in view, the family history of these cases shows us little of importance bearing out these particular contentions.

From the foregoing, it will be readily understood that we do not attach much importance to the fact of there being a family tubercular history in these cases; nor do we think that the existence of such a history lends any, but the most trifling support, to the view of a tubercular origin for the affection.

* Indeed, one of us was at pains to collect the histories of 45 normal cases, and found no appreciable deviation from the numbers here quoted.

Therefore, whilst we contend that we have no proof of tubercular origin in heredity, we do not deny that a tendency to it, or a special vulnerability of the lung, occurs in the descendants of those affected.

Sometimes there is found, in the family histories of these patients, a **tendency to chronic, or, more rarely, acute rheumatism.** This is the more interesting on account of the fact that some observers have found valvular murmurs in a large proportion of their cases. On the other hand, there are some who have altogether failed to demonstrate cardiac complications, with any degree of frequency, and this last remark applies to the series of cases in the sixth chapter. But besides having an effect upon the cardiac complications, a rheumatic diathesis might be supposed to considerably favour the onset of fibrosis in some organ or other of the body, rendered suitable for it. Professor Laycock, in his *Lectures*, has drawn an admirable picture of a rheumatic type of phthisis, and on reading over his description one can hardly escape the belief that he was portraying fibroid disease of the lung. In some cases the chain of events is all but complete. The father or mother of the patient, or perhaps his uncles or aunts, have suffered from rheumatic attacks, which left heart trouble. The patient himself becomes affected with heart disease, following upon rheumatism, and then, soon after, fibroid disease of the lung supervenes.

A family history of **gout, interstitial nephritis,** and **apoplexy** is sometimes to be obtained, though not very often. Still, it is common enough to be looked upon as playing a part in the fibroid process. Now between all these three conditions, and arterial degeneration or fibrosis, there is a close relationship, and interstitial nephritis itself is a fibrosis of the kidney. It may be possible that the fibroid diathesis, above referred to, may so operate as to bring about arterial fibrosis or renal fibrosis in one individual, while in another member of the same family, from some cause or other, it is lung fibrosis that is produced. It will be remembered that one of Laennec's cases ended by an attack of apoplexy. The supposed antagonism between gout and tubercle is well known, although the idea of gout being a prophylactic to tubercle, is now held to be erroneous. Yet one meets with very few cases of tubercle of the lungs giving a history of gout. On the other hand, the gouty or uric acid diathesis seems particularly favourable to the onset of fibroid disease.

Then again, there is sometimes a family history of **syphilis**, but it does not appear to exercise any influence over the fibroid disease.

Lastly, we must consider **cirrhosis of the liver.** There is occasionally evidence of this disease having occurred in the family, but not with any frequency. We have already pointed out that, when occurring in people affected with tubercular phthisis, it does not seem to render the tuberculosis more fibroid and chronic; on the contrary, many, so affected, quickly succumb to a rapid form of phthisis. Recapitulating, then, the various points raised in this account of the family tendencies, we may say:

1. That a family history of tubercle, interstitial nephritis, rheumatism, gout, morbus cordis, syphilis, cirrhosis of the liver, and apoplexy is sometimes present, but that they none of them occur with sufficient frequency to cause them to be regarded as having a direct and special effect in the production of fibroid disease of the lung.

2. That tubercle occurs no oftener than in the family histories of healthy individuals, and for this and other reasons stated may be disregarded.

3. That the other diseases enumerated may, in one generation be manifestations of a fibroid diathesis, whilst in another they give rise to fibroid disease of the lung.

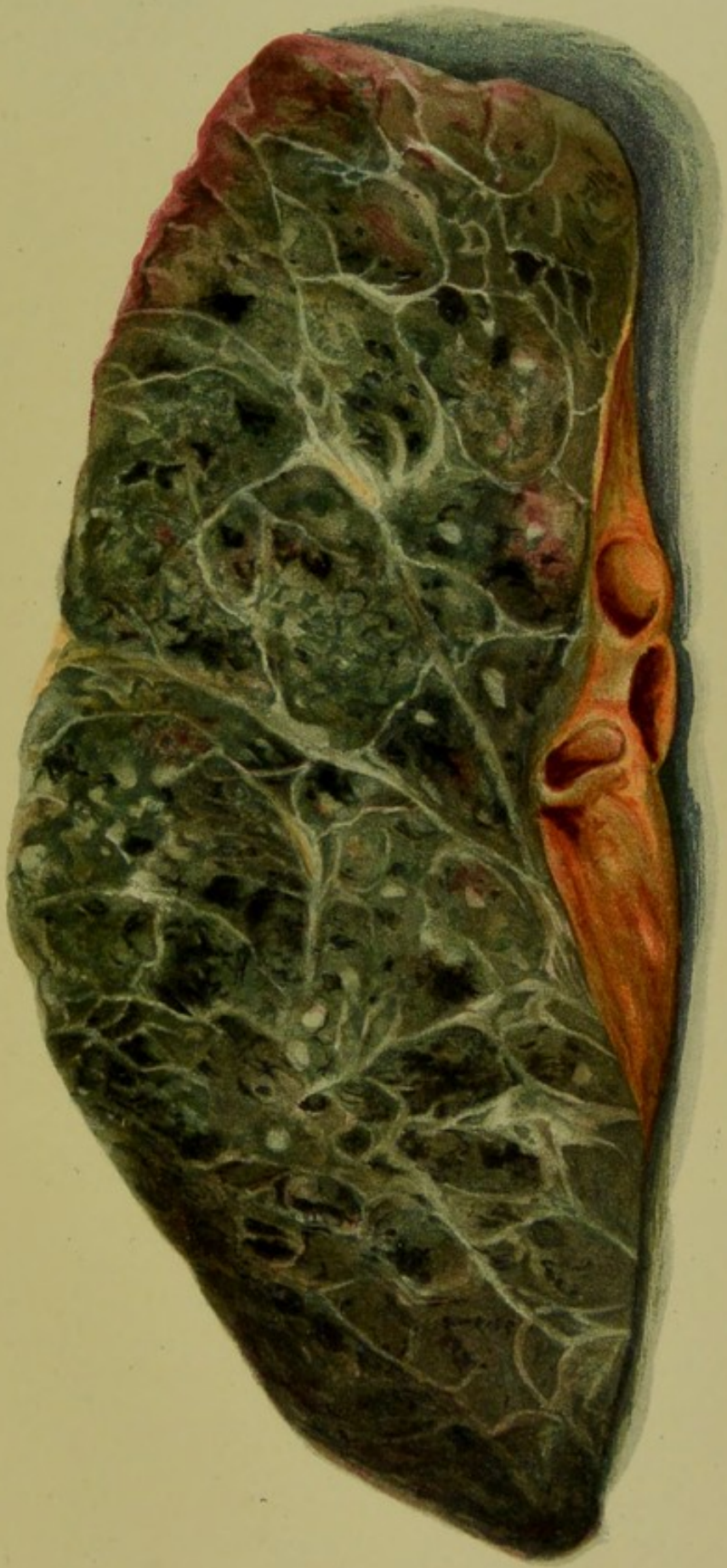
Personal History.—Coming now to the personal history of these cases, there is but little to say beyond what has already been said, when speaking of the ætiology. As a rule, until they become affected with the disease, the health of these patients is remarkably strong, and few of them can be found who give a previous history of ever having been valetudinarians. It is true that not a few of them date the beginning of their disease from a few years after birth, and to such these remarks do not apply. But, in those who are attacked later in life the general statement is, that they were strong, well-grown, able to hold their own in the various walks of life, and were considered as robust and healthy individuals, with the exception of a winter cough. Even after they become victims of the disease they are by no means to be regarded completely as invalids; they are still capable of much bodily activity, and continue to wear the appearance of good health. This is a very different account to that which would have to be written, were one describing the personal history of tuberculous patients, or almost any other pulmonary trouble.

The Mode of Onset of Fibroid Disease is of great importance, for it is a point of diagnostic value enabling us, in many instances, to determine its origin in tuberculosis or otherwise. If we except those cases which owe their origin to prolonged bronchitis, almost every case has some definite beginning. There is generally a history of some acute attack, before which they were in perfect health. These patients can almost always trace the sequence of events up from this attack, and are able to say that, since such and such an illness (whether pneumonia or broncho-pneumonia), they have never been quite well, but have complained of dyspnœa, cough, expectoration, and the like. Now when one has to deal with a case of tuberculosis, the impressions conveyed are very different. There is often no definite onset, nothing to account for the state they have fallen into, and beyond a vague history of cough and weakness gradually increasing, there is no landmark in their past history by which to determine its beginning. Though it must be conceded that sometimes tuberculosis apparently owes its origin to some other lung disease, such as pneumonia or bronchial catarrh, yet this is not the general mode of onset. It is surprising how many of such cases break down when carefully investigated. Further, it may be mentioned that tuberculosis from its onset is quickly progressive, when compared with fibroid disease, which is slow. Where tuberculosis will overrun the greater part of a lung in a term of months, fibroid disease, on the other hand, will take years to attain the same result.

Age.—Coming now to the age at which fibroid disease is most prone to attack people, there exists in published records, and personal experience, much diversity of opinion. As will be gathered from a perusal of the historical account, it is held by some, that it is met with most commonly in the early periods of life, by others, in the later periods. We may state our belief that both periods are liable to the disease, though in which it is more prevalent, it is very difficult to say. The series of cases tabulated in the sixth chapter certainly favours the view, that it is common before the age of thirty, and decidedly rare after it. But we must hesitate to accept these tables as conclusive, for the reasons which are there set forth, more especially when we know that it is the common experience of one of us, to meet with the disease most frequently in the late adult life. This great dis-

PLATE IV.

This drawing shows a typical specimen of Addison's grey induration. The lung is pigmented, and the fibroid tissue in an early state, being seen principally as thin, white, intersecting bands, marking off the lung into areas and planes, and giving it a mottled appearance.



crepancy may be explained, perhaps, on the hypothesis that the cases in the tables are hospital cases, while the others are entirely drawn from private practice.

In fact, while waiting for larger statistics, all that can be said as to the age of patients affected, amounts to this; that many cases are met with in early life, as is shown from statistics, and that it is within the experience of the highest authorities, to come in contact with cases almost exclusively occurring in people advanced in years.

Almost all are agreed that the **duration** of fibroid disease is long—that is to say, the disease lasts years. According to some, an average of twenty or thirty years' duration is not considered too high, others would place it at a lower figure. However that may be, it is certain that the duration of this affection is longer by years than that of any other chronic lung disease, excepting chronic bronchitis.

General Features of Fibroid Phthisis.—Before entering upon a critical account of the signs and symptoms of fibroid disease, it will be of advantage here to give a brief clinical description of its more common features. Patients with fibroid disease generally present themselves giving a history of having had, at some remote period, an acute attack, in the shape of broncho-pneumonia or acute pneumonia, and that, since the attack they have never been quite well in health; or, on the other hand, they may have been subject for many years to a harassing winter cough, which of late, instead of recovering in the summer, has lasted all through the year. The symptoms they generally complain of, are cough of a peculiar paroxysmal kind, coming on most often in the early morning on waking, and sometimes ending by the expulsion of large quantities of phlegm, and not unfrequently, vomiting. They are liable to dyspnœa on the slightest exertion. Sometimes there is a good deal of pain complained of in the affected side, and occasionally, there is an account to be obtained of a fairly profuse hæmoptysis. Patients rarely complain of much emaciation or night sweats. The digestive system is, as a rule, in good working order; at times, however, there is dyspepsia, and sometimes, diarrhœa. Fibroid patients frequently state that, but for the cough, expectoration and dyspnœa, they would think themselves in perfect health. They bear upon their countenances the impress of health. The face, owing to the venous congestion pro-

duced by the lung disease, is turgid, the lips blue, and the eyes dark. There is an almost normal development of muscle and fat; the chest is well-formed and deep. Sometimes white fibroid spots can be noticed in the skin. The fingers are frequently clubbed at the ends, often to an enormous extent.

On inspection of the chest, there can generally be observed greater or less irregular flattening of one side, and impairment of movement during respiration; the shoulder of the affected side droops somewhat, and in some cases, there is a lateral curvature of the spine. On percussion, all degrees of impairment of note can be ascertained, from slight deviation from the normal, to absolute dulness. Generally the disease is most intense over the middle and lower parts of the lung, more rarely at the apex. The tactile vocal fremitus may be either increased or diminished, depending very much upon the thickness of the pleura. On auscultation, there can be heard, bronchial breathing and bronchophony, passing on into pectoriloquy, these phenomena being best marked around the inferior angle of the scapula. In association with these sounds are loud gurgling râles, wheezing rhonchi, and dry, rustling crepitations; the latter being quite superficial. The sound lung is hypertrophied, emphysematous, and encroaches on the affected lung, across the sternum. The heart is almost always drawn to the affected side. Sometimes it may be found situated under the right nipple or in the left axilla, or its position may only slightly deviate from the normal. There is, as a rule, some hypertrophy of the organ, and its beats are slow, infrequent, and measured. A murmur, systolic in rhythm, may often be detected.

The urine of fibroid patients is very apt to contain albumen, often in large quantities, and albuminuria is the rule, after the disease has lasted any length of time. The temperature can hardly ever be observed above normal, and the expectoration contains no tubercle bacilli. This condition of things, with care, may last years without any worse concomitant, and death may not take place until old age; when this event happens it is due generally either to tubercle, capillary bronchitis, or heart and renal troubles.

Symptoms.—We now pass to a critical analysis of the signs and symptoms, presented by those suffering from pure fibroid disease of the lungs.

Cough.—This symptom is one which is nearly always present in a greater or less degree, usually also, with the history that it has

been troublesome for a very long time, often for years, perhaps even for a lifetime. It is worse at times, especially, of course, when the patient has contracted some fresh catarrh, but does not show a great tendency to get worse in winter; and, what is a more marked feature still, is that it does not leave the patient even in the summer. In some cases there is nothing in the cough (apart from its duration) which is at all characteristic. In most cases, however, it is more or less distinctly paroxysmal, worse in the mornings; indeed, the patients are sometimes almost free at all other times. In such cases the morning fit of coughing is very violent and prolonged. Attended at first with no expectoration, the efforts become more and more violent; the face becomes livid, the patient almost suffocated, epistaxis not unfrequently occurs, and at last the expectoration comes in a gush, but often, not until the cough has culminated in vomiting; and in many cases it would appear as though it were voided, more by the action of vomiting than coughing.

Expectoration.—It is evident that at different times, even in the same case, the expectoration must present very different characters. Thus, one finds it occasionally almost absent, or at any rate very scanty, watery or viscid, hardly, if at all, purulent; indeed, simply white, frothy mucus. At other times, it may be muco-purulent, purulent or sanious, varying in colour, according to its consistence, from a greenish or yellow to a reddish-brown, purple, or almost black appearance; at the same time being either quite sweet, of a stale, sickly, or intensely fœtid odour. When voided in large amounts, and indeed often at other times, its heaviness, stale, sickly odour and want of aëration are sometimes very characteristic.

Microscopically examined, the sputum may perhaps present only a few catarrhal cells and mucous granules, with a few pus corpuscles. In other cases, it may be crowded with pus cells, blood discs and catarrhal cells. Elastic tissue may, not unfrequently, be found. Elastic tissue, when present, occurs in two main distinct forms; either in mere cords, strings, or leashes of fibres, or in the form of elastic areolæ.* In the former case they are undoubtedly derived from the submucous tissue of the bronchial mucous

* In a well-known clinical manual, there is given an engraving of leashes of elastic fibres, as indicative of the existence of a cavity in the lung. But such long leashes of elastic tissue do not exist in the pulmonary alveoli, while they are not unfrequently found in the sputa of severe chronic bronchitis.

membrane, in the latter from disintegrating alveoli. As might be expected, the sputa are often found crowded with putrefactive organisms, staining well with methyl blue; but though cases were often under close observation for years, in none of those, which have been chosen as non-tubercular, were there ever any tubercle bacilli found.

Dyspnœa.—The severity of this symptom varies very much in different cases. It must be remembered also, that in some cases the adjustment of the system to the diminished extent of respiratory area, is so far complete, that sometimes, the shortness and frequency of breathing, always present at such times, is not painfully felt by the patient, who will declare that he has no difficulty of breathing, although its shortness and frequency are obvious to the physician. It is always more evident on exertion, and indeed may be present only at such times; but as a wider and wider area of lung-tissue becomes implicated, so does the difficulty of breathing become more and more urgent; until, towards the close, even when at rest, the breathing may be noticed to be shallow and hurried, or even laboured, and the slightest exertion will, in such cases, bring on such a paroxysm of dyspnœa, that the sufferer becomes deeply cyanosed, and looks as though about to die. Moreover, if a patient, a certain portion of whose lung is implicated in fibroid disease, should contract acute trouble, in the shape of bronchitis, or a more or less limited patch of pneumonia, then the consequent further narrowing of his respiratory area renders the dyspnœa most urgent indeed. This complicating bronchitis or pneumonia by no means unfrequently occurs, and the patient then dies of asphyxia with extraordinary rapidity.

Cyanosis.—The presence of this sign depends upon the urgency and duration of the dyspnœa. There may be none; but not unfrequently, where the difficulty of breathing has not yet given rise to actual cyanosis, there may be observed a congested, turgid or swollen appearance of the face, begotten of the prolonged dyspnœa, and the violent straining, caused by the distressing and often paroxysmal cough, added to the direct obstruction to the return of blood from the head and neck, consequent upon the impaired flow through the lungs. This sign is often present, and may give the patient a deceptively robust, or even bloated, appearance. Of the same nature is the clubbing of the fingers, which occurred in 75 per cent. of our cases. It was by no means a

constant sign, being often absent in the best marked cases; and, as it is present in so many other chronic affections, but little reliance can be placed upon it. It is, however, interesting as it shows the result of chronic embarrassment of circulation. The toes are often similarly affected.

Epistaxis.—This sign is intimately associated with the chronic congestion of face above mentioned. It is fairly common, more especially among the younger patients, and is most apt to occur during a paroxysm of coughing. We saw no reason to regard it as having any connection with renal disease.

Hæmoptysis.—This symptom, it will be seen from the tables, occurred in 15½ per cent. of our cases. When one remembers the extreme vascularity, so often presented by the newly formed tissue, the common occurrence of erosions, ulcerations, or a villous condition of the bronchial mucous membrane, with the occasional presence of ulcerating cavities; and, further, when one bears in mind that these conditions are frequently associated with prolonged and exceedingly violent fits of coughing, one is not surprised that this symptom should be of so frequent occurrence. It tends to be often repeated in the same case, and may vary in amount, from only a few streaks to a very considerable loss of blood; and, in rare cases, may be so profuse as to directly and immediately cause the death of the patient.

When one takes into consideration the extreme violence of the cough, and the state of chronic congestion of the whole of the respiratory tract, one has little doubt that, in some cases, the blood comes from the throat. Still less doubt is there that, in the majority of cases, it comes from the bronchial tubes, which, in nearly all cases, present numerous points from which hæmorrhage is only too likely to arise. Whereas, when cavities exist, either extending from, or formed independently of, the tubes; and, as sometimes occurs, their walls are ragged and sloughy, and the excavation is rapidly spreading, then the probability of a large vessel being opened becomes at once imminent, and would result in such a profuse hæmoptysis as to endanger the life of the patient.

Energy and Bodily Vigour.—No one, who has had an opportunity of comparing a number of cases of fibroid disease with those of ordinary pulmonary tuberculosis, can help being forcibly impressed with the striking contrast, existing between them, in this respect. In the cases of fibroid disease, the patients are almost invariably in

active employment; both mentally, and physically, they are vigorous; and, cut off as they often are, by the supervention of some more or less acute trouble, they most frequently remain occupied mentally, or are even engaged in laborious manual labour, up to within a short time of their death. On the other hand, the lack of energy and extreme and progressive bodily enfeeblement, so characteristic of the tubercular affection, must have been remarked by all.

Fever, Sweating, and Emaciation.—These are certainly not symptoms of pure fibroid disease, except under unusual or exceptional circumstances. It is, however, necessary to mention them, as their absence goes far in accounting for the retained mental and bodily vigour, to which attention has just been drawn. Fever does occasionally occur, as, for instance, when a patient becomes the subject of some more or less severe fresh trouble, bronchitis and the like; but as a rule we may expect a normal temperature.

Sweating occurs under similar circumstances, and indeed, in most cases, the skin acts rather freely, perhaps taking on some of the work of the damaged lung; but in no case have we seen the excessive drenching night sweat, so often occurring in tubercular cases.

Emaciation is conspicuously absent, the patients often being quite fat. Towards the close of the case, however, it does sometimes occur; at first slowly, but, as the disease extends, and the dyspnœa becomes more and more urgent, the patient becomes restless and disturbed, does not take his food, and the wasting is more rapid.

Gastro-intestinal Disturbances.—Sometimes it has been noticed, in advanced cases, that there was considerable functional derangement of the stomach. Vomiting is often present, consequent upon the violent cough, and sometimes the vomit is tinged with blood, or this symptom may occur independently of coughing. The stomach is always much displaced in left-sided disease, and also generally, considerably dilated. At the same time, it is conceivable that there may often be a chronic congestion of the organ, consequent upon the back pressure, caused by the impeded flow of blood through the lungs. These considerations fully explain the dyspepsia, vomiting, and slight hæmatemesis occasionally noted by us.

Diarrhœa is often mentioned as of common occurrence in this disease. Certainly the cases we have collected did not verify this. It does, undoubtedly, sometimes occur, caused by chronic congestion of the intestines, or actual erosions of the mucous mem-

brane. But, as far as one can gather from recorded cases, by far the most common cause of this complication is some septic poisoning, begotten of the retention of quantities of foul and stinking secretion in the bronchial tubes; or the death of small portions of the lungs—pulmonary gangrene—giving rise, in either case, to symptoms of general septic poisoning, in the majority associated with diarrhœa, more or less profuse. One of us holds, however, that although diarrhœa may not be common among the younger patients, it is of common occurrence amongst the older ones, and in long-standing cases.

Œdema not unfrequently occurs. It may be found in both the upper and lower extremities, or either alone. When occurring in the lower extremities, it is generally late in its appearance, *i.e.*, when the disease has become very extensive in the lungs, or the general health is giving way. In such cases, it may be accounted for by the back pressure in the venous system, due to the interrupted flow through the lungs, and also, by the direct obstruction to the return of blood from the lower extremities, from implication of the vena cava as it passes through the thorax to the heart, caused by the matting together of the mediastinal tissues. Further, the displacement of the heart far to one or the other side, may, in some cases, prove an impediment to the free entry of blood from the vena cava. All these conditions may exert some influence in causing œdema, but the fact remains that, it is rarely present until late in the course of the disease, and when the patient's powers are rapidly failing.

In some rare cases, the lymphatics and veins, coming from the upper extremity, are implicated in the contraction of the lung, and œdema, which in some case is enormous, occurs of the whole arm corresponding to the diseased lung. In these cases there is direct obstruction to the venous and lymphatic flow; the œdema may begin early, and tends to become more and more marked.

Albuminuria and Renal Disease.—Albuminuria was present in 20 per cent. of our cases. It will be seen, therefore, that in those we have collected, it is not of common occurrence. In the instances in which it was found, it seemed that, other things being equal, it most often appeared when the disease had lasted a long time, and was more likely to be present in those cases occurring in older, than younger individuals. Another point that came out was that, the albuminuria was not constant, at times wholly disappearing

in cases in which it had been previously discovered. It is the experience of one of us, who has dealt chiefly with fibroid disease in middle and advanced life, that albuminuria is almost always present, at least intermittently, after two years' duration of the malady. So that, it may be present in a large number of cases at certain times. As far as we could determine, when the patient was suffering from a fresh catarrh, and the breathing and circulation were more than ordinarily embarrassed, so the albumen would appear; and as he recovered his former state of health, so it would disappear. We came to the conclusion that when albuminuria was present, it was due to chronic passive congestion, and this was borne out by the fact that, at the autopsies of our cases, two of whom had albuminuria during life, we could find no structural disease of the kidneys at all, but they were somewhat enlarged and deeply congested. Two of us think, therefore, that if structural renal disease, apart from congestion, be present, it is simply an accidental occurrence, not caused by, and in no way associated with, the pulmonary trouble.

Having reviewed the general signs and symptoms associated with, and dependent upon the state of the lung, we will now turn to the consideration of the physical signs in the chest, upon which we depend for the diagnosis.

Physical Signs.—In order to have a correct picture before us, we will proceed in the ordinary way in which physical examination is made. In order that the physical signs may be quite definite, we will assume that the case is already considerably advanced, and that one lung only is the seat of disease.

Inspection.—By this means alone, very much may be learnt as to the nature of the case. Thus, it will be seen that the chest is deformed. One side appears smaller than the other, the corresponding shoulder is dropped, the ribs are twisted downwards and inwards, the intercostal spaces more or less retracted, and the spine is pulled towards the affected side. The impulse of the heart may generally be seen displaced from its normal position towards the affected side. All these conditions have been fully discussed in the consideration of the "displacement of viscera," and it is only necessary here, to refer to what has been said there. If now, the patient be asked to draw a deep breath, the affected side will be observed to remain almost motionless, whilst on the opposite, the movement is excessive, the chest there expanding

to an unusual degree, and the shoulder being lifted high up, whereby the contrast between the two sides is remarkably accentuated.

Percussion.—From this we learn that there is dulness, more or less extensive, resistant, and complete, over the affected (contracted) side. Moreover, we notice that this impaired resonance is nearly always at, or towards the base; the apex often remaining free, being sometimes even hyper-resonant. Sometimes the chest gives a dull note from base to apex, both anteriorly and posteriorly; but usually the resonance is impaired throughout, the impairment being more and more marked, until, at the base, especially posteriorly, absolute dulness is reached. On the opposite, least affected side, if the disease (as is usual) is of one side only, an unusually good note may be obtained—indeed, it is hyper-resonant; and, not unfrequently, this hyper-resonance may be traced transgressing the middle line, both along the sternum in front, and the spine behind. Percussion also teaches us that the area of cardiac dulness is, in the majority of cases, enlarged, and displaced towards one side or the other to a degree which varies in different cases.

Further, in left-sided cases, we may frequently elicit the characteristic note of the stomach, as high as the third rib in front; and in right-sided cases, the liver is often so pulled up that its dulness does not reach the costal arch, and would give the idea that it was contracted, did we not bear in mind the displacement; more especially as its dulness cannot, as a rule, be distinguished from that of the lung above it. Sometimes, however, we find, still from percussion, that both the liver and splenic dulness are increased, and in the case of the liver, the enlargement may be very considerable, being due, as we know from *post-mortem* examination, to chronic congestion and fatty degeneration.

Palpation.—By this we at once detect the displacement of the heart, and we gain a certain knowledge as to the character of its beat, which is slow and measured, regular and forcible, quite undisturbed by the gross changes going on around it, and showing a marked contrast to the weak beat of a tubercular case. Then, with regard to tactile vocal fremitus, one finds that, in the majority of cases, it is increased, generally more so at the middle and lower parts of the lung and posteriorly, or in the axillary region. Not at all unfrequently, however, it is diminished, and in a few cases altogether absent. Of course this is on the diseased side, the

tactile vocal fremitus remaining normal on the sound, or least affected side, or is sometimes slightly exaggerated.

Auscultation.—Over the dull area the inspiratory and expiratory sounds are more or less exaggerated, so as to give rise to harsh, bronchial or cavernous breath sounds, the intensity varying in different cases. This is the case in by far the greater majority of instances; sometimes, however, the breath sounds are diminished, and in a very few almost absent. In those cases in which there is bronchial or cavernous breathing, the vocal resonance is also much increased, bronchophony, or even pectoriloquy, being well-marked. On the other hand, in those in which the breath sounds are diminished or almost absent, the vocal resonance is likewise less, or more often absent. In a few instances, although the breath sounds seemed distinctly exaggerated, the vocal resonance showed no such increase. The cavernous sounds, above mentioned, showed a considerable uniformity in the position over which they were best heard. Thus, in almost every case, they were found at the middle and lower parts of the lung; and, further, they were most frequently heard around the inferior angle of the scapula, very rarely reaching so high as its spine. The top, or middle of the axilla was perhaps the place next in frequency. Occasionally these sounds were heard in the mid-scapular region, but, as has been said above, rarely at the apex, although, the apical affection was occasionally observed, alone or in connection with disease lower down; so that it must not be concluded that the base of the lung is the only seat of this disease. Indeed, the lower middle part is the most common situation for its commencement, and thence it spreads downwards before it passes upwards, so that, usually, the lower half of the lung becomes early affected.

Adventitious Sounds.—Scattered over the greater part of the lung, but more numerous, and heard more distinctly at those points where cavernous sounds were heard, were rhonchi, coarse, bubbling crepitations, pleuritic creakings and frictions, and often a peculiar squeaking, sucking sound, difficult to describe, but not easily forgotten when once heard. Of these sounds, the bubbling, and often metallic-sounding crepitations were the most constant, being almost invariably heard. Most often they were associated with pleuritic creakings; and the peculiar sucking sound, last mentioned, was also often heard, and appeared to us the only sign which was of itself peculiar to the condition. It is, perhaps, here

worthy of notice, that fine crepitations are rarely heard in these cases; the crepitations almost invariably heard, as has just been remarked, being of the subcrepitant variety, moist and bubbling.

If the opposite lung remained free from disease, as was the rule, the signs to be heard over it were, chiefly, somewhat exaggerated breath sounds, puerile in their intensity. In those instances in which, in addition to the disease in one lung, there was general catarrh; rhonchi and râles were also to be heard over the otherwise sound side, and in some cases, even when there was no evidence of such a complication, an occasional rhonchus, with a few crepitations could sometimes be heard, more especially at the base of the sound lung. It was considered that these sounds were conducted from the diseased to the sound side.

The Heart.—With regard to the heart, we learn by auscultation more accurately than by any other means, the character of its beat. The regularity, slowness, and steadiness of its action have been before emphasised. It was further remarked that there seemed, in many cases, a rather longer interval between the first and second sounds than normal. Occasionally, there was heard a systolic bruit over the pulmonary area; and, in one case, fairly well-marked systolic bruits, were to be heard, at many points over the diseased lung.

To summarise these various points:

On Inspection.—Deformity, dropped shoulder, twisted ribs and spine, displaced heart's impulse, and deficient or absent movement during respiration, were to be seen.

On Percussion.—Impaired resonance, or more or less absolute dulness—as a rule, at the middle and lower parts—on the diseased side, with hyper-resonance on the sound side. There was sometimes also hyper-resonance of the apex on the diseased side. Increased area and abnormal position of cardiac dulness. Recognition of stomach note high up in the left-sided disease, displaced liver dulness in right-sided disease.

By Palpation.—Displaced impulse of heart. Increased, diminished, or absent tactile vocal fremitus on the diseased side, the same being well-marked on the opposite. Pleuritic friction or râles also felt.

By Auscultation.—*On the Diseased Side*, breath sounds increased to bronchial, or even cavernous, breathing; or diminished, or almost absent. Vocal resonance increased to bronchophony or pectoriloquy, or diminished, or even entirely absent. Coarse, bubbling, and metallic crepitations, rhonchi, pleuritic creakings and frictions, and peculiar squeaking, sucking sound.

Heart's beat heard to be forcible and measured, with apparently a longer interval between the first and second sound than normal.

Occasional systolic bruit heard at pulmonary orifice, and also, in one case, the same to be heard at various points over the diseased lung.

On the Sound Side.—Puerile breathing and a few rhonchi and râles, probably conducted from the diseased side.

The interpretation of these various signs is generally quite simple; the difficulties will be discussed when we come to speak of diagnosis. It will be noted that there is no sign, with the exception of the characteristic sucking sound mentioned, combined with the peculiar action of the heart, which can be fairly considered to be distinctive of this condition, but it is by the assemblage and association of many of them that, we are enabled to arrive at a just conclusion as to the exact state of the lung.

Thus, the dulness associated with bronchial breathing, and the cavernous sounds generally, point to the infiltration and consolidation of the lung, associated with excavation or bronchiectasis, or both. The deformity of the chest, the lack of movement on respiration, the extension of the sound lung over to the diseased side, and the general displacement of organs towards the faulty lung, point strongly to the contracted state of the diseased organ. So far all is quite simple. But in some cases the breath and voice sounds are diminished, or nearly absent, to hand and ear. These signs being still associated with dulness and immobility, point to fluid or a thickened pleura, and here, the contraction of the chest and displacement of organs *towards* the dull side, save us from mistaking it for fluid. It will be noticed, too, that the thickened pleura, so often present, annuls the voice sounds sooner than the breath sounds, the latter sometimes even remaining exaggerated, while the former are diminished.

These adventitious sounds are common to many other conditions, and need no explanation, except the peculiar sucking sound. This occurred during inspiration, and seemed to us to be due to the slow sucking of air and viscid fluid through engorged, rigid, and probably dilated tubes, the slow movement being produced by the tardy and difficult expansion of the indurated lung.

The systolic bruit at the pulmonary orifice may be explained by the altered position of the heart, or the contraction of mediastinal tissues around the root of the pulmonary artery, causing some slight obstruction to the flow of blood through it. The similar bruit heard over the lung might be due to the conduction of this

bruit, or to contractions causing similar obstructive bruits in various branches of the pulmonary artery.

Diagnosis.—We now turn to the question of diagnosis, and although, as has been said, the interpretation of the signs is, as a rule, simple enough, yet, not unfrequently, cases arise in which there is the utmost difficulty in arriving at a definite opinion as to the condition. The various other troubles likely to be mistaken for this disease, are: ordinary pulmonary tuberculosis, fluid in the chest, pneumonia, aneurysm and morbid growths, simple thickened pleura, pulmonary collapse, simple bronchiectasis, and, lastly, the result of old empyemata.

The diagnosis between pure fibroid, and ordinary pulmonary tuberculosis will be fully discussed in a subsequent chapter, when we come to consider the tubercular variety of this disease.

Fluid in the Chest.—Cases of fibroid disease are not unfrequently mistaken for pleurisy with effusion. This would rarely occur, if the past history and the physical signs were carefully weighed. For the fibroid case will usually give a history of many years' duration, whereas that of fluid in the pleura is generally short. But the most important point of all is that, the viscera are all displaced *to* the affected side in fibroid disease, whilst this displacement takes place in the opposite direction in the case of effusion. Moreover, "shifting dulness" may often be obtained in cases of effusion, but never in those of fibroid. In some cases, however, the fluid is located, and no "shifting dulness" can be elicited; or, in cases of either fluid or fibroid, the heart may be fixed by adhesions, so that it cannot be pushed by the one, or pulled by the other, out of its normal position. Then the most important aid as to where the heart is beating, is taken from us. We have, however, other considerations which will almost always enable us to come to a just conclusion. It must be remembered that, in fibroid disease, the displacement of the viscera is complementary, and when there is but little displacement, there will be marked contraction of the chest wall. This is very important in the diagnosis of these difficult cases. Generally, the amount of falling in of the chest, in the case of fibroid disease, is appreciable to the eye, while there is often an undue fulness in the case of fluid, so that a diagnosis may be at once arrived at. If, however, there is any doubt about it, the measuring tape will almost always reveal a considerable difference between the two sides; in the case of fibroid, the side diseased

being less, in that of effusion being greater, than the sound side. Then, too, the percussion note in fibroid disease is usually not so absolutely dull as in the case of effusion, and the voice sounds are never ægophonic, although they are sometimes absent, as has been stated above.

The supreme difficulty in arriving at a correct diagnosis between fibroid disease and pleuritic effusion, occurs in those rare cases in which a pleuritic effusion is subsiding, when the lungs are not expanding, and when, therefore, there may be retraction of the side, with some slight traction of organs towards the affected side, and to some degree or other, those signs which are supposed to be characteristic of fibroid disease.

It is almost needless to remark that, there would never be any difficulty, if the usual signs of a fibroid lung were always present, (such as the cavernous signs, for instance), and it can hardly ever happen that, after a careful review of the signs and symptoms, a diagnosis cannot be made between effusion and fibroid disease. If there should still be a doubt, however, the aspirating needle will at once set it at rest.

Pneumonia.—There can hardly ever be any real difficulty in distinguishing between fibroid disease and pneumonia, the history and the temperature in the vast majority of cases, being quite sufficient to settle the diagnosis. Sometimes, however, there may be some difficulty, and no doubt cases, of even well-marked fibroid disease have occasionally been mistaken for pneumonia. If a case of pneumonia be seen for the first time after the crisis has been passed, or has not resolved till some time after the usual period for resolution, or, on the other hand, if a case of fibroid disease, by contracting a fresh catarrh, develops fever, and shows many of the other signs and symptoms of an acute pneumonia, then a doubt may arise. A careful examination into the history of the case, and the position of the displaced organs, and contraction of the chest in fibroid disease, will, however, invariably serve to distinguish it from the acute affection.

There are cases of recent pneumonia in which the exudation has occurred with such rapidity, and to such an extent, that the substance ceases to vibrate with the moving column of air, and neither tubular breathing nor vocal fremitus are present. In such cases, doubtless, differential diagnosis would be difficult, although not impossible under a critical clinical analysis.

Aneurysm and Morbid Growths.—The signs and symptoms of fibroid disease of the lung, intra-thoracic growths, and aneurysm are often very alike. Thus, we may have in all, a history of cough with expectoration, and not unfrequently hæmoptysis. On examination, we may find dulness, weakened or nearly absent breath sounds, a certain amount of contraction of the chest wall, and in the case of growth, as well as fibroid disease, the heart may be slightly drawn to the dull side.

It will be seen, therefore, that there may be a considerable resemblance, in some cases, between these affections. This is more especially so at first sight, but rarely will it be impossible to distinguish between them, if the doubtful case be watched for a time.

In the first place, as regards the history: fibroid disease may have lasted very many years; aneurysm rarely has a long history, and the course of morbid growths is even still more rapid. The tender age of many of the fibroid cases, may sometimes prove of service in guiding the diagnosis. Then, the part of the chest commonly presenting the signs, differs in the three affections, fibroid disease usually selecting the middle or lower part of the lung, morbid growths the middle and upper; whereas, aneurysm is not uncommonly situated centrally, and at the upper part, though aneurysms of the thoracic aorta often approximate very closely to the position of the fibroid affection. Next, the contraction of the chest is often excessive in fibroid disease, is never anything but slight in the case of morbid growth, and both in this and in aneurysm, there usually is expansion instead of contraction. With regard to dulness, it is often most absolute and "wooden-like" over a growth, rarely so marked over a fibroid lung; breath and voice sounds are, not unfrequently, quite absent in the case of a growth, but rarely, if ever, are they entirely absent over a fibroid lung.

The displacement of organs and parts often helps us materially, for, in the case of fibroid disease, it is generally well-marked towards the dull side, whereas, in the other two, it is the greatest exception for there to be any displacement to that side, and usually, there is movement in the opposite direction.

Further, in the case of pressure symptoms, it is but rarely we get them from fibroid disease. True, occasionally, there is œdema of one arm, but, as far as we know, there has been no case recorded, and we have only seen one, where the radial pulses were unequal.

Distended veins over the chest wall are rare in fibroid, but not at all an uncommon accompaniment of morbid growth. Inequality of the pupils has never been recorded in fibroid disease, and in only two cases has there been paralysis of the vocal cord. In one of these it was due to a local affection, in the shape of ulceration, and in the other, to the implication of the recurrent laryngeal nerve on the right side, as it curved beneath the subclavian artery in the extremely thickened pleura at the apex of the lung. On the other hand, the frequency of the occurrence of pressure symptoms in the other two affections, especially perhaps aneurysm, is well known. Finally, the aneurysm may be accompanied by pulsation, a distinctive bruit, or some well-marked cardiac or general arterial disease, whereas, the course of the morbid growth, with its occasional typical sputum, possible occurrence of sanious or other pleuritic effusion, progressive emaciation and cachexia, and occasional supervention of secondary malignant deposits elsewhere, will always serve to correct any error, there may have been at first, at no very distant date.

Thickened Pleura.—So often is the pleura very much thickened in cases of fibroid disease, and when it is so, the breath and voice sounds are generally very much diminished by it, that, in this respect at any rate, the two affections are precisely similar. Moreover, not unfrequently, the thickened pleura, by extension into the lung, will eventually give rise to fibroid disease. Therefore, from a diagnostic point of view, it is often only the degree of contraction of the lung, and the result of this contraction on the chest wall, and the neighbouring viscera, which enable us to distinguish between the two. This is usually quite sufficient, more especially, as the extreme contraction which is often found in fibroid disease of pleuritic origin, generally readily distinguishes such a case from one of simply thickened pleura.

Pulmonary Collapse.—This will give rise to dulness, more or less marked, diminished or absent respiratory murmur, absent tactile fremitus, and vocal resonance; and, when large areas are affected, even cause falling in of the chest wall. But there are never any cavernous sounds, the contraction of the chest is usually not marked, and displacement of surrounding organs almost never takes place.

Simple Bronchiectasis.—This affection may occur without fibrosis, in the sense in which we have been using the term, just as

advanced conditions of fibroid disease may exist without any bronchial dilatations. Inasmuch as we know that fibroid disease will cause bronchiectasis, and bronchiectasis, in its turn will give rise to fibrosis, we should expect to find the two affections often combined. Nevertheless, they do occur separately. The points, chiefly to be relied upon in distinguishing a simple bronchiectasis from one occurring with fibroid disease, are the absence of physical signs of contraction or consolidation of the lung. There will be the history of chronic cough, often of the typical paroxysmal nature, attended by the occasional presence of characteristic fœtid sputum, voided in gushes, with the presence of cavernous sounds over the region of the dilated tube, and with, perhaps, a limited amount of dulness. But, there will be no contraction of chest, or displacement of organs, and dulness, if present, will never be anything but slight in amount; points, which will serve to distinguish it effectually from fibroid disease.

The Results of Old Empyemata.—It is not always easy to distinguish the contraction of the chest, associated with a certain amount of thickened pleura and collapsed lung beneath it, which are all that remains of an old purulent effusion into the chest, from a case of fibroid disease. Sometimes, no doubt, there is a certain amount of fibroid induration in these cases, and the differential diagnosis is rarely of very great importance. In the cases of empyemata, where the remaining inflatable lung is more or less healthy, many of the signs, commonly observed over a fibroid lung, will be wanting. For example, there will be no cavernous sounds and no adventitious sounds whatever. There will, however, be more or less impaired resonance, and diminished voice and breath sounds. Further, these signs will be associated with an amount of contraction of the chest, which in some cases is remarkable.

The diagnosis may often be rendered quite simple by obtaining a clear history of the withdrawal of pus, or even by the discovery of the place of its exit, or history of expectoration of the same. But, even without this, we are not often at a loss in coming to a conclusion, as to the true nature of the case. For, although the chest wall has fallen in, there is hardly ever any displacement of the viscera towards the contracted side. On the contrary, in most cases the heart will be found towards the sound side, having been previously pushed there by the purulent effusion; and, as after the pus had gone, the lung could not dilate, so neither could the heart

return to its normal position, both being prevented by adhesions. So, too, the shoulder is dropped in both cases, and the spine is curved. But the curve is commonly in the opposite direction in the two cases, the concavity being towards the contracted chest in the case of old empyemata, the convexity being towards the diseased lung in fibroid cases.

Course, Complications, and Prognosis.—It will be seen that the duration of pure fibroid disease is very many years. Even when death occurs, some complication is often the direct cause, acting on a system whose vitality is somewhat impaired by the chronic trouble, and the congested state and disordered circulation in many organs, which are occasional accompaniments of long-standing fibroid disease. Speaking generally, therefore, in the majority of cases, the prognosis as regards life, and fairly useful life, is distinctly good; but, as the patient is laid open to the supervention of many complications, which materially qualify such a prognosis, each case must be judged for itself. But with all these perils one can still say that, the existence of fibroid disease, need not necessarily interfere with the achievement of the longest duration of life. The commonest complication of all is caused by the onset of fresh catarrh, resulting in more or less general bronchitis. This places the patient sometimes in considerable danger from asphyxia, more especially, if the catarrh should extend to the smallest tubes (capillary bronchitis). It is then that we find the dyspnœa, accompanied by cyanosis, becomes most distressing, and it is then, that we fear the extension of the acute trouble to the alveoli themselves; both on account of the extreme danger to the patient's life, at the present, from the supervention of pneumonia, and because these attacks sometimes result in the further crippling of the diseased lung, in the future.

Some patients are particularly liable to contract these fresh catarrhs, either from some inherent tendency, or, in the case of those who must work, by exposure at their employment under unfavourable conditions. It follows, therefore, that recurrent attacks of bronchitis, with all their attendant dangers, seriously qualify the favourable prognosis which we have given, as ordinarily characteristic of the process.

Occasionally, the fibroid lung becomes infected with tuberculosis, and, considering what a very vulnerable point it is, and what a rich field it presents for the inoculation and cultivation of the bacillus,

it is a matter of wonder that this complication does not occur sooner, and more often, than it does.

The result of this complication is usually immediately marked by the supervention of hectic and sweating, soon followed by wasting and general enfeeblement. The physical signs may not be much altered for a time, though, in some cases the disease tends to spread very rapidly; but almost invariably, sooner or later, and, generally, at no distant date, the opposite lung becomes affected, and at the usual place for tubercular attack—viz., at the apex. In the meantime, tubercle bacilli have generally been detected in the sputum, which otherwise has not materially altered its characters. The future course of the case is generally rapidly downwards, though by no means always so, and the patient may struggle on for some years after the onset of tubercle, though the general course of the disease is much shortened.

Therefore, the discovery of tubercle bacilli in the sputa will render the prognosis much less favourable, and will probably alter the whole complexion of the case, especially if the disease has extended to the opposite apex. But the general state of health must also be taken into account, for a paucity of symptoms, attached to a plethora of physical signs, makes our prognosis comparatively favourable, even in tubercular phthisis.

Gangrene of the Lung.—This is a complication which is fortunately very rare. Its cause, from an anatomical point of view, has been already discussed. It remains, however, to consider its effects upon the existing disease, and the dangers to life, to which its supervention exposes the patient. Generally, during or closely following some fresh catarrh, the symptoms may be noticed to become suddenly altered. The temperature is apt to assume distinct evening rises, to 102° or 103° F., often preceded by a shivering feeling, or even a distinct rigor, to be followed in the morning, by profuse sweating. The expectoration, which previously may have been muco-purulent and odourless, may become fœtid; or, even if fœtid already, the fœtor may become most intense. It may at the same time become tinged with blood—thin, acrid, and watery, or grumous and darkly stained. Later, there may be more distinct, even dangerous, hæmoptysis. Many of the symptoms point to septic poisoning, and, in addition to the fever and sweatings, there may be some slight jaundice, and often diarrhœa of septic origin supervenes. The physical signs may be but slightly altered,

especially at first, but later, there is generally some extension of the dulness; and if the patient should survive, as the slough disintegrates, and is got rid of by expectoration and absorption, we should expect to find the evidence of the cavity thus formed.

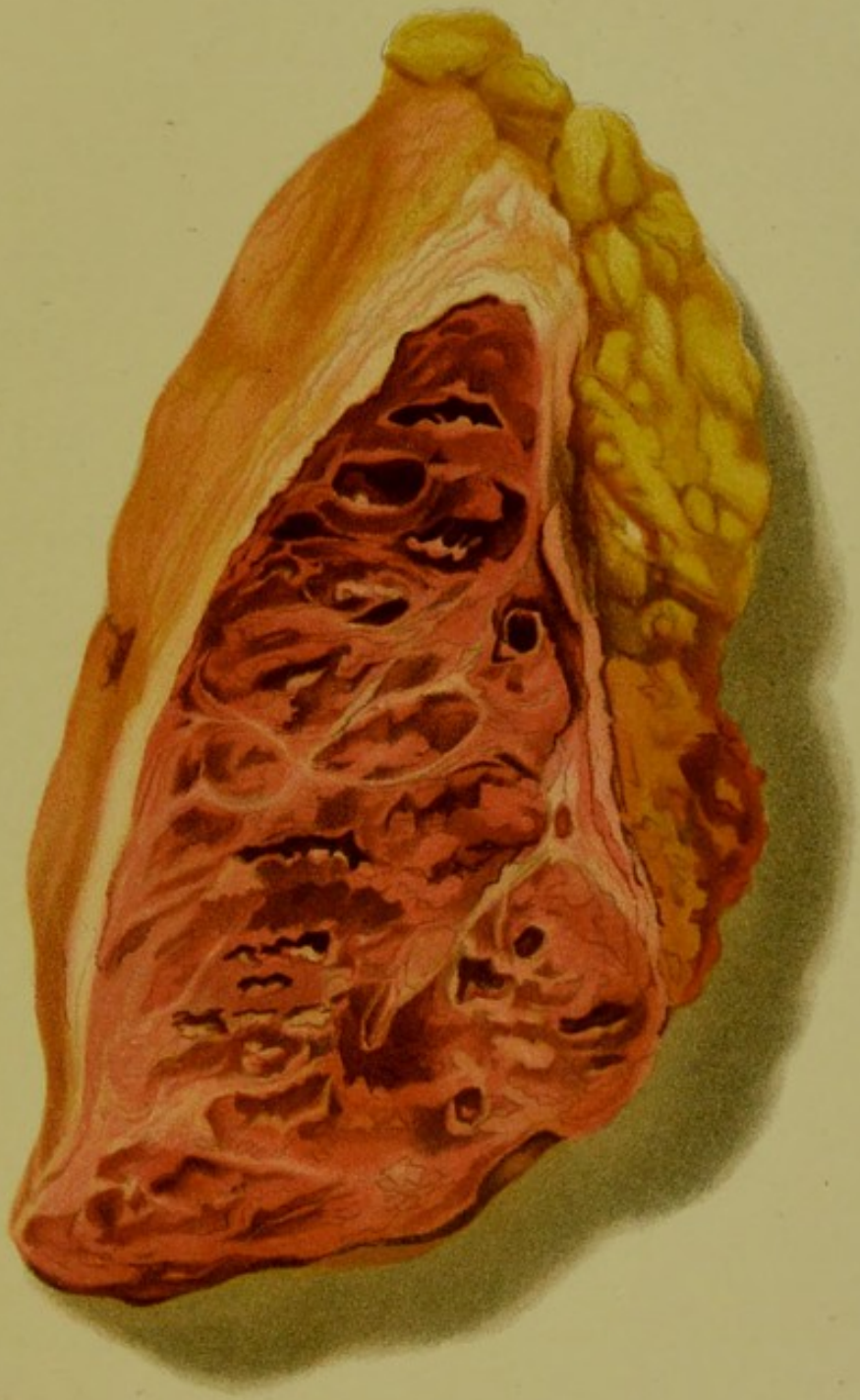
The fatality of gangrene of the lung is very great, few recovering in whom the portion of lung dying is of any considerable size. Death may be caused by the induced septicæmia, or even pyæmia, perhaps, more commonly, by exhaustion. A septic form of pneumonia may be set up in the diseased lung, or in the opposite; or fluid or pus may appear in the opposite pleura, or in the pericardium; or there may be distinct secondary deposits elsewhere.

Fœtid Character of Sputum.—Should the patient recover from the immediate dangers of gangrene, the sputum will often remain fœtid for a considerable time, or continue so, until death. But where there has been no gangrene, there is often considerable fœtor of the expectoration. This condition is begotten of the retention, and consequent decomposition of the sputum in the dilated tubes or in cavities, and is often intensified by more or less extensive ulceration, with the accompanying molecular death of portions of the walls of the bronchial tubes or pulmonary tissue. When this fœtor is constantly present, it must be looked upon as a symptom of bad omen. For, in addition to the deleterious effects on the general health, by the constant absorption into the system of the septic products of the lung, it must be remembered that such is the irritation of this fœtid material, that it may cause ulceration of the tubes, or promote their rapid extension of that process, if it already exists. It may produce small patches of gangrene or a septic form of pneumonia, and will always cause such irritation of the walls of the bronchial tubes that peri-bronchitis will be set up, which will often extend into the lung-substance beyond, and result in the spread of pulmonary induration. On the other hand, many patients with fibroid disease and bronchiectasis, are in the habit of having periodical attacks of aggravated cough and expectoration, and, towards the close of these attacks, fœtid expectoration will appear, continue for a few days, and then disappear, leaving the patient quickly to recover the former level of his health.

Though of nothing like so grave an import as pulmonary gangrene, constant fœtor of the sputum must always influence our prognosis adversely. Patients generally suffer very considerably in

PLATE V.

Is an illustration of fibroid disease of traumatic origin, caused by the perforation of the chest by the shaft of a carriage. The pleura is here enormously thickened, and the lung-substance largely replaced by fibroid tissue, dilated tubes, and irregularly-shaped excavations. A collection of fat is seen around the apical part of the pleura.



general health, and the lung trouble is very apt to progress rather rapidly.

Recurrent hæmoptysis has been mentioned, and as it has a tendency to recur in the same patient, and may be so profuse as to endanger life, it must carefully be borne in mind, when making a prognosis.

As has been said, the prognosis, as a rule, is exceedingly good, and a great many patients escape all the complications mentioned, and live to a good old age, and eventually death may be due to some other cause.

The disease, however, has a tendency to spread, and the longer it lasts, the larger and larger is the area of pulmonary tissue rendered useless. Great is the power of adaptation, but sooner or later the general health fails. Dyspnœa is now more often marked, and so small is the remaining area of useful lung, that the patient may fall an easy prey to catarrh. Or, even without any such complication, œdema is apt to become a more prominent feature, and may attack the lung also, the patient dying rather suddenly at the end; or, diarrhœa may become profuse, sometimes apparently taking the place of the œdema, and then death comes on more gradually from exhaustion.

CLINICAL CASES.

We will now give some cases illustrative of the foregoing account.

Case I.—R. L., aged 17, was admitted into the City of London Hospital for Diseases of the Chest, on May 16th, 1893, under the care of Dr. Thorowgood (to whom we are indebted for permission to reproduce the case), giving the following **history**. When two years old, the patient had measles and whooping-cough, which was followed by an attack of broncho-pneumonia. Since that attack, she has never been free from cough, dyspnœa, and expectoration. She has had frequent attacks of bronchitis, with an accession of her other symptoms, necessitating her admission to hospital. As a child, she has been in the East London Hospital for Children, under the care of Dr. Eustace Smith, on several different occasions. Until three years ago, she has had recurring attacks of hæmoptysis, but never in large quantity. She has complained, from time to time, of night sweating, and pain in the right side, but has never had any emaciation worth speaking of.

Her **family history** presents no features of interest, there being no evidence of tubercle, nor anything which would account for her present state.

Appearance.—On admission, the patient was found to be a fairly well-built young woman; there was a good covering of fat and muscle. The face was broad and the lips cyanosed, the general appearance of the countenance being thick and congested, very much resembling that of a patient with mitral regurgitation. The eyelashes were not unduly long, and the hair was crisp and short. There was but slight clubbing of the fingers. The pulse was regular, full and infrequent, with a certain degree of tension. The temperature was all but normal, and remained so during her stay in the hospital.

Symptoms.—The patient complained of cough, dyspnœa, and expectoration, and, as a rule, but for these symptoms, her health would be described as good. There was evident dyspnœa, the respirations being 24, still more increased on exertion; the alæ nasi were working slightly. The cough had a peculiar character, it was paroxysmal and severe, coming on worst at about 11 A.M., increasing in severity, and often culminating in vomiting. By this severe cough large quantities of expectoration were expelled, thick, lumpy, but never fœtid. The cough and the expectoration, and their periodicity, all suggested the emptying of dilated bronchial tubes. The digestive system was fairly good, the tongue slightly furred, appetite good, bowels regular. There had never been any diarrhœa.

Physical Signs.—**Inspection** of the chest showed great retraction of the right side, and consequent drooping of the right shoulder. The mammæ, however, were almost on a level. The movement was much diminished over the right side, and the whole of that side of the chest had the appearance of being fixed. The measurements of the two sides gave the following results: at level of the sixth rib, left side, 14½ inches; right side, 13 inches; at the level of the nipple, left, 12½; right, 11. The left side seemed unduly prominent, but this was probably due to the great amount of retraction on the right. On palpation, the tactile vibration was increased on the right side, and some rhonchial fremitus could be detected. There was an impaired note on **percussion** at the apex, of the right side, back and front, passing towards the base, into absolute dulness. On **auscultation**, all over the right lung could be heard loud bronchial breathing, attended with coarse crepitations and superficial creak-

ings. On speaking, bronchophony, and at the inferior angle of the right scapula, pectoriloquy, could be clearly distinguished. The left side was slightly contracted at its base behind, and here the note was a little impaired. Over the whole of this lung there were coarse rhonchi and bubbling râles. These latter signs cleared up well during the patient's stay in the hospital. The left lung on percussion was found to extend considerably over the sternum, to the right side.

The Heart.—The apex-beat was in the fourth interspace, on the right side, just below the right nipple. Auscultation and percussion showed that very little of the organ was to the left of the sternum, transposition to the right side having taken place, owing to the retraction of the right lung. The heart's beats were measured, slow, and infrequent; and occasionally, at the apex, could be heard a "whiff" with the first sound. The hypertrophy of the heart could not be made out, but might be inferred from the character of the beat. The liver and spleen were sound and healthy (so far as could be determined).

Urine was 1020, of acid reaction, and showed the constant presence of a faint trace of albumen.

The **sputum** was repeatedly stained for bacilli, but always with negative results.

Case II.—D. H., aged 23, a gardener, has been under observation for six years. He gives the following **history**: When quite young he had whooping-cough and measles, the cough remained after these illnesses, but was not at all severe. When nine years old—that is, fourteen years ago—he was laid up with an acute attack, which was called, at the time congestion of the lungs; since this last attack his cough and expectoration have become aggravated, and have never ceased to trouble him, summer and winter. Since the congestion of the lungs, his dyspnœa has been excessive on the slightest exertion. He has had one or two attacks of hæmoptysis of trifling amount. He has never sweated to any extent, and has uniformly maintained his weight. The cough is of a peculiar character; it is paroxysmal, worse in the morning on waking, and at times brings on vomiting. The expectoration is profuse and lumpy, and has a fœtid odour. He has had one attack of rheumatic fever, but it did not seem to have any effect upon his shortness of breath, which was present long before that. He stated that, but for his cough, dyspnœa and expectoration, he would

consider himself a strong, healthy man. Lately, he has had one or two sharp attacks of epistaxis.

Family History.—Among his relatives, his father died of phthisis, at the age of forty; beyond this there is nothing of note in the family history.

In **appearance**, he is a healthy-looking young man; the face looks congested, and the lips are slightly blue. The finger-tips are markedly bulbous. His work is gardening, but he is quite equal to the demand made upon him by his occupation. On **inspection** of the chest, he seems to be well-nourished, with a normal amount of muscle and fat upon him. The whole of the left side of the chest is very much flattened, and moves hardly at all during the respiratory act. The left shoulder droops perceptibly. Over the whole of the left lung the tactile vibration is increased. On **percussion** of the left lung, the note is found hyper-resonant at the apex, down to the second interspace; from there downward, the note becomes more and more impaired, until at the sixth rib in the nipple line it is absolutely dull. Below this, stomach resonance is detected.

Behind, in the supra-spinous fossa, the note is impaired, and quite dull as the base is reached. On auscultation, all over the impaired area, there are bronchial breath sounds, bronchophony, and at the inferior angle of the scapula pectoriloquy; to these sounds are added loud, coarse, bubbling râles, creaking and fine rustling crepitations.

The **right lung** is hyper-resonant and extends to the left edge of the manubrium sterni, the breath sounds are exaggerated, but otherwise normal. The **heart's** apex-beat is $2\frac{3}{4}$ inches outside the nipple line in the left axilla; the beats are slow, infrequent and measured, and there is a suspicion of a mitral stenotic murmur at the apex. A systolic bruit is detected in the left supra-spinous fossa. The pulse has the same characters as the heart-beats.

The **urine** contains a large quantity of albumen, about one-fifth. This was not detected when in the hospital five years ago. The temperature has always been normal. The expectoration has been stained repeatedly for tubercle bacilli during the last five years, but hitherto none have been found.

Case III.—**History.**—E. Mc——, a female, aged 14 years, was admitted to the City of London Hospital for Diseases of the Chest,

under the care of Dr. Clifford Beale, to whom we are indebted for permission to reproduce the facts of the case here. She came complaining of having had a "chest trouble" since she was sixteen months old, at which time she had whooping-cough. She has never been free from cough and expectoration since that time. On questioning her as to the nature of the cough, one found that it was distinctly paroxysmal in the mornings, was associated with copious expectoration, which came up in gushes, sometimes attended by vomiting. She had never had any hæmoptysis, neither had there been any appreciable amount of wasting at any time.

Present State.—She looked somewhat undergrown for her age, and her face presented a congested, turgid appearance. There was considerable dyspnœa, and she complained of pain in the right side. The fingers were distinctly clubbed. On examination of the chest, it was at once evident that the right side was contracted; the corresponding shoulder dropped, and the whole side remained immovable during respiration. Percussion revealed dulness of the whole of the right side, from apex to base, becoming more absolute as the base was reached. Bronchial breathing, with well-marked pectoriloquy, were to be heard over a considerable area around the inferior angle of the right scapula, and extending up into the mid-scapular region of the same side. These cavernous sounds were accompanied by loud, coarse, bubbling râles, heard also over most of the lower part of the right lung.

The tactile vocal fremitus was considerably increased over the whole of the lower part of the right side. The heart was displaced far to the right, the maximum impulse being seen under the right nipple, in the fourth right intercostal space. There was no bruit.

The temperature remained uniformly normal, and the urine was free from albumen.

Here we have a case of at least twelve years' duration, but in which, although the disease is extensive on the right, there is as yet no disease whatever to be found on the opposite side. The left lung, indeed, presented only the signs of emphysema. Neither has the general health suffered, and nature has so far compensated for the damage done to the lung, that up to the time of writing, there are no other evidences of impeded circulation, beyond the clubbing of the fingers, and the turgid condition

of the face. There is, as yet, no œdema, and no albuminuria. Tubercle bacilli have never been found in the sputum.

Case IV.—A man, aged 22, was admitted, under the care of Dr. Sainsbury, at the City of London Hospital for Diseases of the Chest, who has kindly permitted us to publish the case here. He gave the **history** that he had had whooping-cough at four years of age, and had been ailing, more or less, ever since; but that he had been much worse since eleven years old, at which age he had contracted "inflammation of the lungs," and since which time, he had never at any period been free from cough and expectoration. The expectoration was fœtid, and sometimes attended by hæmoptysis. The cough was worse in the mornings, but otherwise was not particularly characteristic.

On examining the chest, contraction and immobility of the left side were at once apparent, with the usual dropping of the corresponding shoulder. Dulness, most absolute at the base, shaded gradually into impaired resonance at the upper part; but there was no spot on the left side which gave a good note to percussion. Associated with this dulness and contraction, were cavernous sounds, including bronchial breathing, well-marked pectoriloquy and bronchophonic cough, to be heard over the greater part of the lower third of the lung, behind, and in the axilla, but marked best around the inferior angle of the scapula. Over the whole of this lung, except the extreme apex, might be heard bubbling, coarse râles and wheezing rhonchi, which were more copious over the lower half of the lung, and heard best over the areas where cavernous sounds were also marked. In this case there was some evidence of the disease having also implicated the right lung; for over the lower third of that side, the movement seemed lessened, the note of percussion was decidedly impaired behind, the breath sounds were loud and harsh, but could not be called bronchial; and there were numerous rhonchi, and coarse, bubbling râles, to be heard, especially over the area of impaired resonance.

The heart was displaced to the left side, the apex-beat being perceptible one inch outside the nipple line. The area of cardiac dulness was enlarged, probably largely due to the uncovering of the heart, by the retraction of the lung. No bruit was to be heard at any of the orifices, neither was any bruit heard over the lung; whilst the action of the heart was quiet, regular and

vigorous, and peculiarly measured. In this case the fingers showed distinct clubbing, but, though repeatedly examined, no albumen could be found in the urine. There seemed nothing in this case worse than in many others, except the fœtor of the expectoration, which we believe to be always a sign of bad omen. He was, however, subject to recurrent catarrhal attacks, and in one of these, passing on to a severe attack of capillary bronchitis, he died asphyxiated in a few days.

Tubercle bacilli were never discovered in the sputum.

A full account of the *post-mortem* appearances in this case will be found, in the chapter on morbid anatomy and pathology.

CHAPTER V.

TUBERCULO-FIBROID DISEASE—FIBRO-TUBERCULAR DISEASE—TREATMENT.

WE must now speak of that variety of the fibroid process, which we have proposed to call by the name of **Tuberculo-fibroid Disease.** By this designation we mean to imply that form which, in the first instance, arises and runs the course of ordinary chronic tuberculous phthisis, but in which, as time goes on, the fibroid process gains supremacy, the tuberculous manifestations becoming either obsolete or, at any rate, of only secondary importance. When such an occurrence takes place, the case very much resembles pure fibroid disease, though there are not wanting diagnostic signs and symptoms which, when recognised, render the two forms fairly easily distinguishable. A correct diagnosis between these two states is of paramount importance, not only to the physician, but also to the patient, for, the prognosis of the two affections is widely different; the fibroid disease, when pure and simple lasting almost any length of time, but not nearly so long, when complicated with tubercle. On the other hand, the prognosis of tuberculosis, which has become fibroid, is much more hopeful than when it exists without an undue amount of the fibroid material.

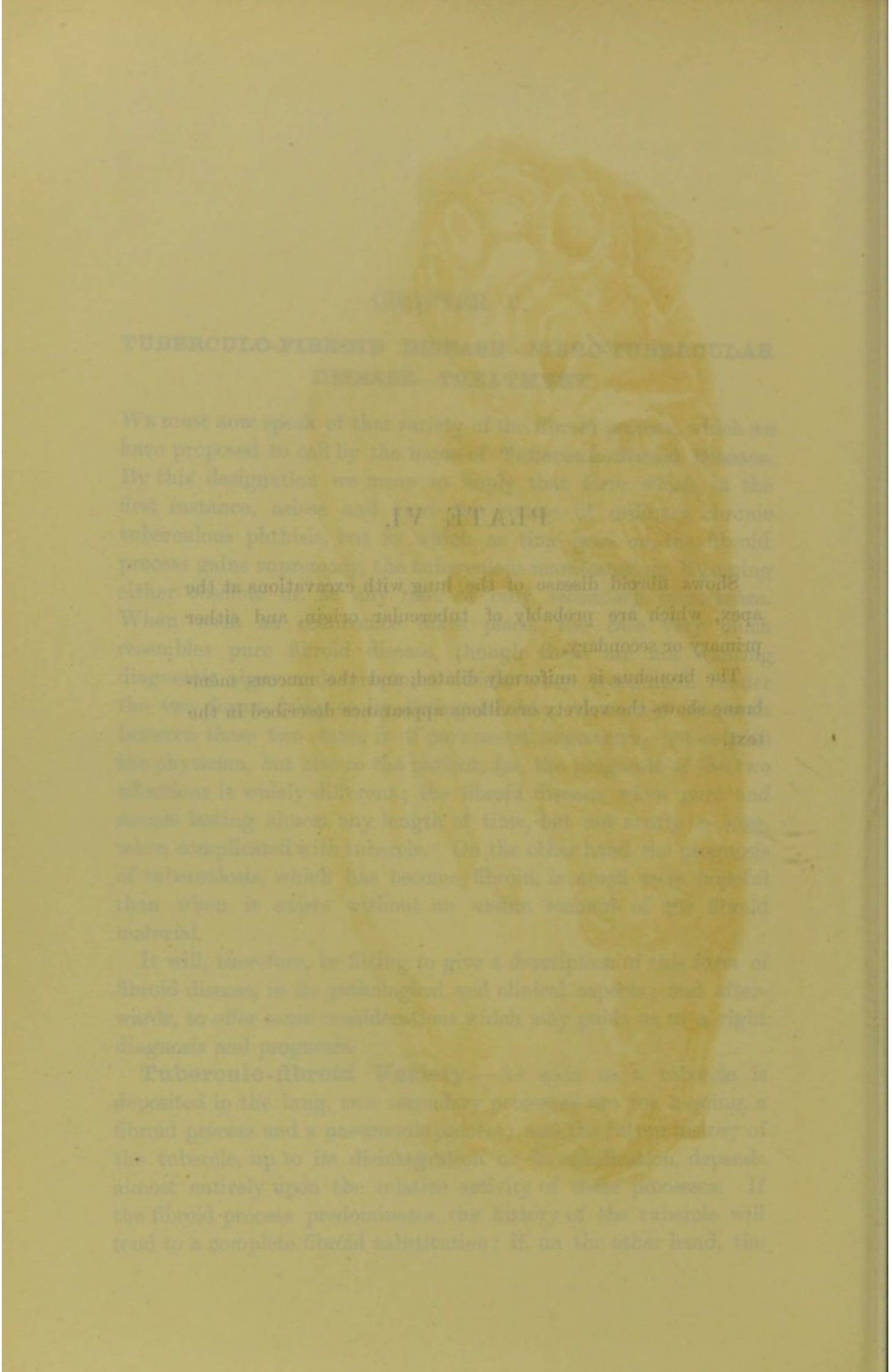
It will, therefore, be fitting to give a description of this form of fibroid disease, in its pathological and clinical aspects; and afterwards, to offer some considerations which may guide us to a right diagnosis and prognosis.

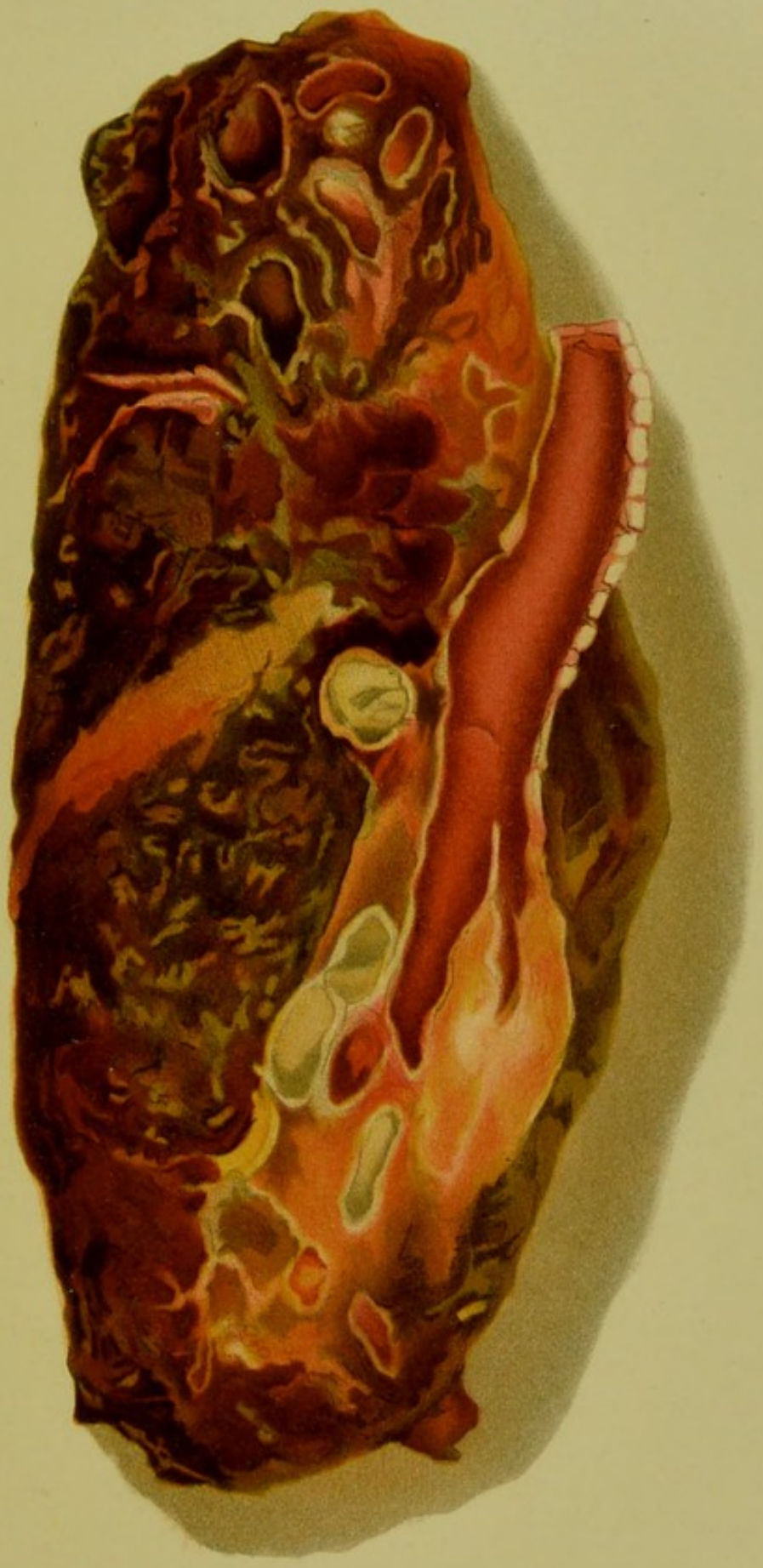
Tuberculo-fibroid Variety.—As soon as a tubercle is deposited in the lung, two secondary processes are set a-going, a fibroid process and a pneumonic process; and the future history of the tubercle, up to its disintegration or its calcification, depends almost entirely upon the relative activity of these processes. If the fibroid process predominates, the history of the tubercle will tend to a complete fibroid substitution: if, on the other hand, the

PLATE VI.

Shows fibroid disease of the lung with excavations at the apex, which are probably of tubercular origin, and either primary or secondary.

The bronchus is uniformly dilated, and the mucous membrane shows the velvety or villous appearance described in the text.





pneumonic process predominates, the history of the tubercle will tend to suppurative or ulcerative disintegration. If we imagine that the lungs are more or less crowded with tubercles, and that the like processes are going on, then we know for certain that, in those cases where the pneumonic process predominates, the course of the disease will be febrile and rapid; and that, in those cases where the secondary processes are fibroid, the future of the case will be non-febrile and slow.

In a case of acute tuberculosis of the lung there is hardly ever time for the fibrosis to assert itself, while, in a chronic case of lung tuberculosis, there is always found a plentiful supply of fibroid tissue. Indeed, the amount of fibroid tissue, in a lung affected with tuberculosis, may be taken as a measure of the prospect of life. But, in all cases of chronic tuberculosis, the fibroid tissue does not so completely gain the upper hand, and overrun the whole lung so extensively as to cause it to simulate pure fibroid disease in an advanced state. By far the greater number of them run their course without, at any time, becoming liable to be mistaken for pure fibroid phthisis, and, perhaps, the reason for this is to be found in the fact that, nearly all cases of chronic pulmonary tuberculosis are slowly progressive; in other words, the tuberculous process, however chronic, is almost always in excess of the fibroid process. Therefore the fibroid process, never completely gaining the upper hand, has never an opportunity of putting upon the disease the impress of signs and symptoms peculiar to it.

To this class of case, the description about to be given will hardly apply. There are, however, a few cases of lung tuberculosis, in which the fibroid processes have obtained such complete supremacy and the tuberculous process has become so insignificant, that they appear, to the casual observer, to be cases of pure fibroid disease of the lung.

There can be no doubt that it is owing to a want of proper distinction between these two sets of cases, that, so frequently, pure fibroid disease of the lung has been assigned to a tuberculous cause.

With these prefatory remarks, let us pass on to give a brief description of tuberculo-fibroid disease, pointing out wherein it differs from the pure fibroid variety. After this it will be well to enter into a critical analysis of the signs and symptoms.

General Description.—A patient, with tuberculo-fibroid

disease, gives a history somewhat as follows. His health is good until, after some slight ailment, he finds he has a cough; this goes on gradually increasing and becoming more and more incessant; at the same time he notices that he is losing flesh, and becomes aware of a perceptible diminution in his strength. The expectoration, which at first was difficult and frothy, is now free and in larger quantity, and he may have hæmoptysis, in larger or smaller amounts; sometimes, indeed, blood-spitting may be the first symptom which attracts attention to his state of health. His appetite soon begins to fail, and a troublesome night sweating may make its appearance. His respiration becomes more and more hurried, and as time goes on, all these symptoms increase in severity, until at last, he seeks medical aid. On examining his chest, it can at once be seen that, so far from its being well-formed, it is shallow in its antero-posterior diameter, and the clavicles stand out prominently from the ribs. The sternum, too, may be depressed below the level of the rib cartilages. The hair and eyelashes may be long and lank, and the fingers slender, long, and tapering. The skin, to the touch, is of a pungent heat. The pulse is quick and frequent, and the contractions of the heart are of the same character. On employing percussion and auscultation, disease of one, but usually of both lungs, can be found, and the physician has no difficulty in pronouncing the disease to be tuberculosis. Tubercle bacilli can be detected in the sputum. Hitherto, there is nothing in the signs and symptoms different from those met with in ordinary tuberculosis, and the case is simply one belonging to that class. After a time, however, varying from a few months to a few years, the case (if it be about to become one of tuberculo-fibroid disease) undergoes a slow, but complete change. The tuberculosis, which had progressed until it had involved a considerable part, or even the whole of one lung, becomes more and more chronic. There is less and less evidence of constitutional disturbance.

The temperature, which before, was always raised in the evening, is now often normal; the sweating becomes less, and the patient regains much of his wonted strength and flesh. If one keeps the case under observation all this time, it will be seen that the affected side becomes more and more flattened, the rib spaces fall in, and the movement becomes appreciably diminished; at the same time, the impairment of note becomes increasingly marked, until absolute dulness, extending over a great part of the lung, can be made out.

The displacement of organs now makes its appearance. The heart is drawn towards the affected side, the stomach may be drawn up, if the disease is left-sided. The lung least diseased becomes affected with compensatory hypertrophy or with emphysema, and extends across the sternum to the most diseased side. If the heart be now examined, often some hypertrophy can be made out, and the beats of the organ are much altered; instead of being quick and frequent, they are slow, measured, and infrequent. The sounds heard over the diseased lung also become gradually altered. In the place of the sharp, fine, moist crepitations which could be noticed when the case was first seen, there are now coarse bubbling râles, rhonchi, and fine superficial rustling crepitations. The bronchial breathing and bronchophony are intense, and often, pectoriloquy can be heard. The fingers may become much clubbed, and sometimes the urine becomes albuminous. Even the countenance can sometimes be observed to wear a congested appearance. The dyspnœa remains very much what it was at first, but the cough and expectoration undergo a change. The cough is now paroxysmal, often bad in the morning on waking, and attended with gushes of expectoration, which suggest the emptying of dilated tubes. After lasting some time in this condition, the patient generally succumbs to a fresh eruption of tubercles in the least affected lung, or to an attack of hæmoptysis. Owing to the altered condition of the blood-vessels, and especially to their diminished elasticity, hæmoptysis is sometimes more prolonged and severe in these cases than in cases of ordinary tuberculosis.

Morbid Anatomy.—On examining the lung after death, the pleura is found to be enormously thickened over the ancient seat of the tuberculosis. There is extensive fibroid formation all through the lung; the bronchial tubes are dilated, but not to such a great extent, as is met with in pure fibroid disease. The lung, as a whole, is much contracted, and the division into lobes is lost. There are, at different situations in the lung, old, dry, puckered, thick-walled cavities, around which the fibrous tissue is amassed in great quantity. Scattered all over the lung, but most thickly at the apex, are cheesy masses of tuberculous matter, which are encapsuled; some of them are even calcareous. These cheesy masses, when examined by appropriate means, are generally found to contain tubercle bacilli. The least affected lung, in addition to being emphysematous, contains cheesy nodules of tuberculous matter,

and often an eruption of fresh, grey granulations, which has been the cause of death. The bronchial glands are enlarged, and may contain caseous or calcareous foci of tuberculous matter. The heart is not so large as in the pure variety of fibroid disease. The liver is often enlarged and congested, and the same may be said of the kidneys and spleen. Lastly, the intestines often show typical tuberculous ulcers.

To a casual eye, this tuberculo-fibroid state much resembles, in its signs and symptoms, pure fibroid disease. A closer and more critical inspection will, however, bring to light points of difference, which make it difficult to mistake it for the latter affection. Let us now inquire what these distinguishing features are.

In the first place, the **onset** of the disease is unlike that of pure fibroid disease. There is no definite illness or cause, such as broncho-pneumonia, acute pneumonia, or prolonged bronchitis, from which the patient is able to date the beginning of the disease. It begins insidiously, as it were, with no starting-point, and it is only after it has gone on for some little time, that the patient becomes aware of his condition. It is not contended that tuberculosis never begins after such attacks as those mentioned above; but it is submitted that, beyond a chill or some other slight affection, it is the exception, rather than the rule, for tuberculosis to be definitely traceable to one of the above pulmonary diseases.

Secondly.—From its **symptoms and signs**; for a long time the disease presents all the features of ordinary tuberculous phthisis, and there is nothing in the case to lend any support to the idea that it will ultimately become fibrotic. Whereas, in pure fibroid disease, from the very first onset, there is a train of signs and symptoms peculiar to it, which, when carefully investigated, has little in common with ordinary tuberculosis of the lungs (except that which belongs to the impairment of the organ in relation to sound).

Thirdly.—The presence of **tubercle bacilli** in the expectoration, forms an important point of distinction. In tuberculo-fibroid disease, they may be said to be always present in the expectoration, both when the disease is simply tubercular, and often long afterwards, when the disease has taken on a fibroid form, and is then tuberculo-fibroid. Sometimes, however, bacilli cannot be detected in very old tuberculo-fibroid lungs, yet they can be found if, after death, the caseous matter be examined for them. This is very

different from what obtains in pure fibroid disease of the lung Here there are never, at any time during its course, bacilli in the expectoration, unless it be when tuberculosis has become grafted upon it.

Fourthly.—The **temperature**, in tuberculo-fibroid disease, is different from that in pure fibroid. In the former affection, when the disease is beginning and before the fibroid process has sprung up, the temperature is always raised in the evening to one or two degrees, or even more, above the normal. When the tuberculosis has become fibroid, there is a recession of the temperature towards the normal line; but it does not remain so for long. Every now and then, owing to the still smouldering tuberculosis, there is an exacerbation of fever, which, in its turn, gives way to quiescence. When all the tubercle has become obsolescent, and provided there is none in progress in the sound lung, then the temperature may always be normal; but such a fortunate ending as obsolescence of all the tubercle is rare. In pure fibroid disease, unless there be some complication, the temperature is persistently normal.

Fifthly.—The **emaciation and sweating** are different in character and amount, from those met with in pure fibroid disease. At first, as would be supposed, they are considerable, but, as the fibroid transformation takes place, they become much less, yet, except in very rare instances, they never cease entirely, being liable every now and then to break out for a time, and then, almost as quickly, subside. How different this is to pure fibroid disease will be readily understood when it is remembered that, in this affection, emaciation and true night sweating, at any time in its course, are the exceptions rather than the rules.

Sixthly.—The **aspect** of the patient offers a striking point of contrast between the two diseases. A case of tuberculo-fibroid disease, however fibroid it may become, always bears, in the general aspect of the patient, evidence of its tubercular origin. The cast of countenance is not of that heavy kind, so often observable in the pure fibroid variety. The lips are not thick and congested, the complexion has not the muddy look, the hair remains lank, and the eyelashes long. The whole appearance of the face suggests tubercle and frail health. It is true that one does meet with tuberculo-fibroid disease, in which the fibroid characters are somewhat clearly marked in the face, but never, so far as we are aware, to the extent of its being mistaken for pure fibroid disease.

Seventhly.—Almost the same may be said of the **contour of the chest.** In tuberculo-fibroid disease it is narrow, the shoulders are high, the sternum depressed, and the clavicles stand out prominently. It is never so deep and well-formed, and so well covered with fat and muscle, as in pure fibroid disease.

Eighthly.—On studying the evidence afforded by the **physical signs**, we meet with important differences between the two affections. First, as to the position of the disease. It will be remembered that the common position for pure fibroid disease was the middle and lower lobes, or at least, that that was the place in which the process started, and that the apex became affected, if at all, much later on in the disease. But, in tuberculo-fibroid disease, the part of the lung, to become earliest affected, is almost always the apex; it is only latterly, and when the fibroid process is fully developed, that the rest of the lung becomes involved. And even then, the tubercular process will be found most advanced at the apex, whilst the fibroid process will be found most advanced at or towards the base. On examining a lung, the most extensive disorganisation is at the apex; the flattening and impairment of movement is most conspicuous there, the other parts of the chest, in this respect, being not nearly so much affected. The dulness, too, is often most extreme here, while the rest of the lung may only be impaired on percussion. Then, again, auscultation reveals phenomena, somewhat different from those met with in pure fibroid disease. In this latter affection the various auscultatory signs, such as coarse gurgling râles, fine rustling crepitations, bronchial breathing, &c., are, as a rule, heard best at the middle and lower parts of the lung; whereas, in tuberculo-fibroid disease they are most advanced, and heard with greatest intensity at the apex. Indeed, as has already been noticed in pure fibroid disease, the apex may be quite free, and present to the ear and finger only emphysema. It is questionable if the contraction, in tuberculo-fibroid lungs, ever reaches such an extreme degree as is seen in the pure variety. Although this may help in determining the true nature of a large number of cases, yet one must not forget that pure fibroid disease does sometimes affect the apices primarily, and tuberculo-fibroid the bases of the lung primarily, though these are, perhaps, the exceptions which prove the rule. In these somewhat anomalous cases, the course the disease has taken, together with an independent consideration of the signs and symptoms, will serve best to

guide one to a correct judgment. Indeed, one might almost attempt to lay down the rule that, with a few exceptions, tuberculo-fibroid disease begins at or near the apex and spreads downwards, while pure fibroid disease begins at the middle and lower parts of the lung, and from thence spreads upwards.

Very often, on examining the opposite lung, a deposit of recent tubercle can be found at the apex, and it is this which commonly causes the exacerbation of hectic, to which patients are liable.

Ninthly.—The character of **the heart and its pulsations** will throw some light upon the diagnosis. When speaking of pure fibroid disease, we pointed out that the heart is often hypertrophied in one or more of its chambers. In tuberculo-fibroid disease there may, it is true, be some hypertrophy, but never to the extent to which it reaches in the pure affection. Then too, although, by the onset of fibrosis upon tuberculosis, the heart-beats may be changed from the quick frequent beat to the slow, infrequent, and measured beat, yet this change is never so complete and marked as in the pure variety. On palpating and auscultating, the cardiac contractions seem to retain some of the phenomena characteristic of the antecedent tubercular condition. If the heart does quiet down, so to speak, the change is not permanent, but it is liable to return to its first state when an exacerbation of tubercle sets in. The displacement of the heart shows little variation from that which can be noted in pure fibroid disease. The displacement may, perhaps, not be quite so great on account of the smaller amount of fibroid tissue in the lung producing contraction.

Tenthly.—The presence of **albumen** does not appear to be such a constant accompaniment of the affection, as in pure fibroid disease.

Eleventhly.—In tuberculo-fibroid disease, the **expectoration**, in addition to containing bacilli, would be more likely to have a larger quantity of areolar elastic tissue than pure fibroid disease, and this would be accounted for by the fact of greater lung destruction being produced by the disorganising effects of the tuberculosis. The clubbing of the fingers will not assist us much in forming an opinion as to the case; for there does not seem to be much difference in the proclivity of the two affections to this feature.

Twelfthly, and lastly, the **duration** of tuberculo-fibroid disease is by no means the same as in pure fibroid disease. We have already seen that, in this latter affection, the duration is extremely long, and

bears no relation to the duration of ordinary tuberculous phthisis. While the duration of pure fibroid disease may be measured in periods of ten, twenty, thirty years, or even longer, the outside limit, except in exceptional cases, of the duration of chronic tubercular phthisis, may be put at five or six years without committing any great error. But neither of these approximate durations would be at all correct if applied to tuberculo-fibroid disease. It does not last nearly so long as pure fibroid disease, and is yet much longer than chronic pulmonary tuberculosis. In fact, from what we have been able to observe of this particular affection, we should be of opinion that the average duration was from eight to nine years. This duration depends very much upon two factors: (1) the amount of fibrosis in the lung; (2) the amount of active tuberculosis in the opposite lung. But more will be said about this when speaking of the prognosis.

The age at which tuberculo-fibroid disease occurs, does not follow exactly in the same lines as the pure variety, for it does not seem, so often, to have its beginning in early life, as does the latter affection. In adult life there is practically no difference between the two diseases, so far as regards the age at which they are apt to supervene. It is submitted, then, that these points will, in some measure, enable one to determine as to the true nature of the affection, whether it be a case of pure fibroid disease, or of fibroid disease which began as an ordinary tuberculosis.

The **Prognosis** of this variety has still to be dealt with. The mere fact of this disease being involved in the tuberculous process, would render the prognosis more unfavourable, than if it were entirely separate from it. On this account, the prospect of life in people with tuberculo-fibroid disease, is not so good as in those in whom there is only the fibroid process. Although the tubercular part of the disease may have become obsolescent, though the lung affected may have become entirely fibroid, yet there is always a risk of a sudden outbreak of tubercle in the least affected lung. When this takes place and spreads to any extent, then the prognosis is very grave, for the only lung, capable of carrying on the function of respiration, is endangered. Therefore, it is of great importance in giving a prognosis, to possess as accurate information as possible as to the state of the apex of the least affected lung. With no active disease in this quarter and quiescence elsewhere, the prognosis is not bad; but with active mischief at the apex of the least affected lung,

a very guarded opinion must be given. Then, the amount of fibrosis, as shown by the contraction of the chest, will assist us. Indeed, the more evidence there is of fibrosis, the better the prognosis; but, on the other hand, the more the case approximates to the tubercular type, and the more active tuberculosis there is, the worse must be the prognosis. The common, indeed, almost the only way, in which these cases end fatally, is by the tubercle gaining the upper hand in the fibroid lung, and by fresh outbreaks in the hitherto but little affected lung.

Fibro-tubercular Variety.

We have reserved this name for those cases which, commencing as pure fibroid, have, at a subsequent period, become complicated by the addition of tubercle. They will be found, therefore, to present many of the points which distinguish cases of pure fibroid disease; added to which, and often considerably modified thereby, will be many of the signs and symptoms which characterise the ordinary tubercular pulmonary affections.

All chronic cases of pulmonary tuberculosis will also have a more or less considerable amount of the fibroid element. Sometimes they run a fibroid course, but are, nevertheless, tubercular from the beginning. These cases, however, only present one side of the picture of possible tubercular and fibroid combinations; and the fibro-tubercular variety, commencing as pure fibroid, with the subsequent addition of tubercle, shows the other side of that picture. We must therefore distinguish this variety, both clinically and anatomically, from cases of pure fibroid on the one hand, and, on the other, from those cases of tubercle which have run a fibroid course. It follows from their origin in pure fibroid disease, that these cases will give the history characteristic of that affection, with the addition that, for the last few weeks or months, as the case may be, they have become suddenly and rapidly worse in every way. Sometimes the cases will have been under observation previously, as instances of pure fibroid disease, during which time, they presented none of the symptoms of tubercular trouble; and, though frequently examined, their expectoration never contained any tubercle bacilli. We have had the opportunity, on one or two occasions, of watching the onset of this tubercular complication. Such patients change with regard to their symptoms almost immediately. In fact, the alteration in the whole complexion of the case, is most

marked and sudden. They become feverish at night, and often sweat abundantly towards the morning. They rapidly fail in flesh, strength and colour, the loss of flesh and strength being often extremely marked. The cough not unfrequently becomes much more distressing and the expectoration more copious, and the shortness and difficulty of breathing, even to them, a more marked feature.

On examination, the physical signs of a contracted, and in part consolidated lung, will be discovered; evidences of the old, fibroid, and formerly uncomplicated lung trouble. To these signs, perhaps, for a short time nothing fresh will be added, except that the moist sounds will become more numerous and extensive. And the symptoms, so different in their character and intensity, added to the examination of the sputa, and the valuable information obtained by the use of the thermometer, and altogether the suddenness and rapidity of the onset and course of these grave changes, will be the points of chief importance in making a diagnosis. Before long, however, the signs of disease will be found extending. Thus, at one or both of the apices of the lungs, signs will appear; first, usually crepitations, soon followed by the signs of consolidation and excavation. The apex of the diseased, or the hitherto sound, lung may be the first to become infected, or the apices may become, more or less, simultaneously diseased. The important point to remember is, that when tubercle infects a case of fibroid disease, whether it primarily attacks the diseased or the hitherto healthy lung, it almost invariably chooses the usual seat for tubercular affections of that organ, and appears first at the apex.

The examination of the sputum, which, as has been said, has often become more copious, will reveal the presence of tubercle bacilli. These generally make their appearance early, with the first change in the symptoms and the onset of hectic fever; often before any alteration in the character, or extent of the physical signs can be noted.

The future course, towards more or less extensive tubercular disease of the lungs, and it may be of other organs, attended by progressive emaciation and enfeeblement, is now often exceedingly rapid, the patient not unfrequently succumbing to his malady in a few months. Although this is the rule, occasionally a case may be met with in which it is not so, and we have now under observa-

tion one patient, who, for the last twelve months has had symptoms of tubercular infection, with the appearance of bacilli in the sputum; but the physical signs are, as yet, but little altered, and he is still able to do some work, although much weaker than formerly.

It is most important to remember in this connection, that, in almost every case the disease, now having become tubercular, will extend to the other lung: it will no longer remain limited to one side. The case, just mentioned above, is somewhat an exception to this rule; for, though the onset of tubercular trouble dates a year back, the disease still remains limited to one side; but we confidently expect to detect changes in the opposite apex before another year has passed, as in all the other cases. It is quite possible that they may be already there, but not in sufficient amount to be detected by physical signs; only to be revealed, as is often the case, at the autopsy.

It will be seen, therefore, that the distinguishing points between this variety and cases of pure fibroid, may be summarised as follows:

1. The history of wasting, sweating, fever and progressive bodily enfeeblement, of somewhat sudden onset, and rapidly increasing.
2. Although there will be the physical signs of a contracted fibroid lung, there will be, in addition, the signs of tubercular affection at one or other of the apices, and not long after the onset of tubercular infection, the opposite lung will almost invariably become the seat of similar disease, usually at the apex.
3. Examination of the sputum will reveal the presence of tubercle bacilli.

To distinguish this variety from cases of tubercular disease of the lungs, which have run a fibroid course, and in which there is a considerable amount of fibroid tissue formation, with consequent contraction of the lung, we have many points which aid us.

The fibro-tubercular cases, having commenced as pure fibroid, have the long history pointing to that affection, during which time, the constitutional disturbance was but slight, with a later history of the sudden onset of more or less profound constitutional symptoms. For the same reason, in examples of this variety, the physical signs will usually be found most abundant in the middle and lower thirds of the lung, whilst in those of tubercle, the upper third of the lung will almost invariably be found the most advanced in disease.

It is on this account that, in cases of tubercle running a fibroid

course, there is rarely any great amount of displacement of neighbouring parts. For contraction of the upper part of a lung has but little power to produce these displacements, when compared with similar contractions of the lower portions.

In the later stages of the two affections, when both lungs have become more or less extensively diseased, it is sometimes difficult to say which was the initial affection, and so large a proportion of the disease is now tubercular, even in the fibro-tubercular cases, that the distinction is not so important. A careful inquiry into the personal history of the patient will often, however, be of the greatest service. With regard to the morbid anatomy in these cases, much of what has been said with reference to the condition, found in cases of pure fibroid, holds true here also. Thus the fibroid thickening of the pleura and interlobular tissue is alike in both. Indeed, having begun as cases of pure fibroid, they will still present those changes which characterise that affection. In those parts, such as the apices, which are more distinctly tubercular, it is not common to find those changes in the alveoli, so characteristic of the pure fibroid affection. The fibroid tissue is often very abundant, and more often completely replaces the true pulmonary tissue, than is the case in pure fibroid disease; and when such portions are treated with acetic acid, actual loss of tissue is much more frequently found than in that affection. Caseous material is also more abundant in the tubercular portions, and cavities are more numerous.

In most cases, in some part, tubercles will be visible to the naked eye, and it will rarely be difficult to find in sections, some more or less typical tubercular formation. Although it is sometimes exceedingly difficult to demonstrate the presence of tubercle bacilli, one can do so, by repeatedly examining fresh scrapings from cavities, or caseous portions, or by appropriately staining sections taken from the caseating areas, in which they are more readily found.

It will be gathered from this description that, however closely the primary and secondary affections may have become combined, in their effect upon the general organism; however close that combination may appear in the lung taken as whole; it will be generally a simple matter to pick out portions, which display under the microscope, more or less typically, the changes characteristic of either the one or the other affection. Thus, evidences of

tubercle may be absent, or only found with great difficulty, in the more purely fibroid portions; whilst fibroid changes may be but slight in amount, in those portions which show the most distinct evidences of tubercle.

Treatment.

This part of the subject may be most naturally discussed under two heads—namely, that which is directed to the maintenance or improvement of the general health, and that which deals, more directly, with the malady from which the patient is suffering, or any complications which may arise in its course. But more important than these considerations is that of **possible prevention.** We know many of the causes, and if we can remove the patient as far as possible from the risk to which they expose him, we shall probably prevent many cases of fibroid disease. Such causes as the inhalations of irritating dust have only to be mentioned; the chronic bronchial and pulmonary irritation will often be arrested, before it has given rise to any great amount of fibroid induration, if the patient can be persuaded to leave his unhealthy employment, his unsuitable surroundings, and the vicious climate in which, perhaps, he dwells. But there are other causes, the effect of which, we believe may in a considerable measure be lessened by the careful treatment of the initial trouble. These are pneumonia, chronic bronchitis, and pleurisy, and the abuse of alcohol. Of all the causes of fibroid diseases of the lung, pneumonia we believe to be the most preventible. Our own cases teach us the enormous proportion which is traceable to this cause, amongst the poor, exposed, as they often are, to the neglect of this not unfrequently obscure disease; whilst amongst the well-to-do, this cause of the affection is quite insignificant. These facts speak most strongly, and we would therefore urge the importance of the careful treatment of these cases, especially of children who have contracted some inflammation of the lungs after whooping-cough or measles. In order that complete resolution may take place, avoidance of fresh catarrh, and a supply of good, nourishing food are most important.

The neglect of a pleurisy (and when there is little or no fluid effusion it is especially likely to be overlooked) may also be another cause of a, more or less extensive, fibroid disease of the lung. Appropriate treatment, especially with rest, will, not unfre-

quently, be attended with good results. And it is important to remember that, if the patient be allowed to go about his ordinary avocations with a dry pleurisy, the irritation of the constant movement will materially increase the amount of the exudation of coagulable lymph, which will result in fibroid thickening of the pleura, often with extension into the lung-substance of bands of fibroid tissue. Chronic bronchitis, later in life, is not unfrequently followed by indurative changes in the lung, and it is of great importance, whether these have begun or not, to endeavour to check the constant recurrence of this winter trouble. It will often be necessary to use the greatest care to prevent it, some individuals apparently being prone to develop bronchial catarrh on the slightest exposure to cold, or even from over-exertion. Where it seems impossible to do this otherwise, it may be necessary to advise a change, during the winter, to a warmer climate, or to get the patient to change an employment, necessitating constant exposure, for one more protected. However this may be, it is important to treat this bronchitis carefully, and, if possible, insist on there being no fresh exposure until the attack has been cured.

When the fibroid change is established, the treatment of the general health is of the first importance, to which may be added that for the prevention of its extension, and of the supervention of complications.

Occupation and Diet.—In the first place, the patient is, as a rule, far better when employed actively in as healthy an occupation as circumstances will permit; and the energy of mind and body, which is usually well maintained, in most cases admits of this being carried out. It is very important that they should have as nourishing a diet as possible; indeed, without advocating intemperance in the way of either meat or drink, we are convinced that such patients do better when really well fed, and a liberal allowance of stimulants seems, in many cases, to be beneficial, if not necessary.

Precautions against Cold.—The great importance of guarding against cold in every shape or form is undoubted. We should therefore ensure that our patients are always warmly clad, and, as far as possible, protected from the danger of contracting catarrhal attacks. Warm clothing is in another way important, in order to keep the skin acting well, and so relieve, as much as possible, congestion of internal organs. This may be further helped by the

regular use of warm bathing and friction to the skin. In a word, the patient should be taught to live the healthiest life that is possible for him, a liberal diet being necessary, the protection from cold with free action of the skin being of vital importance.

Climate.—Change of employment has been mentioned as sometimes necessary; and in some cases, where the winter invariably brings with it severe attacks of bronchial trouble, it may be necessary to advise a change of climate, and urge the patient to pass the winter in a warmer place. It must be remembered that these patients, with their considerably narrowed pulmonary area, do not usually stand high altitudes well, and there is no necessity to try them in this way; they do well almost anywhere in a warm and equable climate. Indeed, if there be albuminuria, patients affected with fibroid disease almost invariably do badly in high altitudes.

Complications.—Various complications may arise which will need special attention.

1. **Bronchitis and Pneumonia.**—The treatment of the catarrhal attacks, which often complicate this disease, differs but little from that which would be employed in ordinary cases. Great care must be taken to guard against fresh exposure, and every effort should be made to shorten the duration of the malady as much as possible; to prevent it spreading to the alveoli and setting up a catarrhal pneumonia.

2. **Hæmorrhage.**—This complication is of such frequent occurrence, that a few words as to its treatment is not misplaced here. It is not common for the hæmorrhage to assume alarming proportions, and it is generally quite sufficient to put the patient in a recumbent position, completely at rest, to ensure its soon ceasing. Sometimes, however, it will be necessary to employ the use of heat to the feet by hot bottles or a footbath, at the same time keeping the body as cool as possible, giving ice to suck, and arranging the diet, so that nothing hot or stimulating shall be taken while the hæmorrhage lasts. These means will, almost invariably, produce a satisfactory result, especially when aided by small doses of morphia or opium to further quiet the circulation, procure cessation of all cough, and produce sleep, during which the bleeding will often cease. The use of astringents is, as far as we have seen, likely to do more harm than good. In some cases, where the hæmorrhage is more profuse, though the patient is not suffering

from the loss of blood which he can ill afford to lose, but where there is danger of the blood doing damage, by its mechanical presence in the lung, the propriety of taking blood from the patient's arm, in order to check the internal hæmorrhage, must be considered. In some cases, we can well believe that this measure, heroic as it may at first seem, may prove of use, may even save the life of the patient. For undoubtedly deaths do occur, due to hæmoptysis alone, and most frequently the actual cause of death is asphyxia, and not loss of blood.

3. **Bronchiectasis and Fœtid Sputum.**—Bronchiectasis is so commonly present in such cases, and its environment of contracted fibroid lung when taken into consideration, together with the fact that it is often not one, but many tubes which are affected, we believe that it is inadvisable to use operative measures for its treatment. But in some cases, retention of sputum or ulceration of the walls of the tube, one or both, have given rise to fœtor of the expectoration. Indeed, when fœtor occurs from any cause, it is most important to try and lessen this condition, both on account of the general health of the patient, which is invariably seriously affected by it, and also on account of the effect of the irritating properties of the decomposing matter on the lung itself. For this purpose antiseptic inhalations are often beneficial, such as benzoin, thymol, or creosote. The internal administration of creosote or iodoform is sometimes attended with good results. Still better, as striking more directly at the root of the evil, is the method recommended by Dr. Grainger Stewart, of intra-laryngeal injection of some antiseptic preparation. The method is fraught with no danger, and is attended by usually rapid improvement.

4. **Gangrene or Abscess of the Lung.**—These most serious complications, fortunately of rare occurrence, will need the most careful treatment. As in cases of fœtid expectoration, arising simply from retained and decomposing sputa, antiseptics are of the greatest use. They may be employed as inhalations or given internally, but by far the most efficacious way is by the intra-laryngeal injection mentioned above. The antiseptic treatment is of the greatest importance, since it is directed immediately to the root of the evil, and since it lessens the danger and amount of septic absorption. The general treatment for septic cases must also be carried out. Thus, the patient must be put on as stimulating a diet as possible, the free use of stimulants is often most

PLATE VII.

The drawing represents the right lung of a boy aged 4 years 10 months. He had been under the care of Dr. Thomas Barlow, to whom we are indebted for the drawing.

At the autopsy there were some old adhesions of the right pleura, but they were easily broken down, and were not thick.

The right lung as a whole was not much contracted in bulk. In the upper lobe there was dilatation and ulceration of the bronchi, with much surrounding fibrosis, which in places was as firm as fibro-cartilage. The invasion of the fibrosis was not from the pleura, but was evidently peri-bronchial. In the middle and lower lobes there was also dilatation of the bronchi, with less amount of surrounding fibrosis. There was no obvious tubercle and no caseation.

The left lung showed a slight degree of fibrosis in the lower lobe.

The trachea and bronchi were inflamed, and contained an excess of muco-pus.

The larynx was natural.

The heart presented some dilatation of the right ventricle, but no valvular lesion or congenital defects could be found.

The pericardium was natural.

There was a little firm caseous tubercle in the cortex of both kidneys.

The other abdominal viscera were healthy.

The brain membranes were normal, and were easily stripped from the brain. The convolutions of both hemispheres presented extensive gliomatosis, and there were some massive upgrowths, probably gliomatous, from one corpus striatum.

In the left retina were several firm, circumscribed, rounded masses, without any trace of caseation, which, according to Mr. Nettleship, were an unusual form of fibro-plastic tumour.

The case had been under the observation of Dr. Barlow for more than four years.

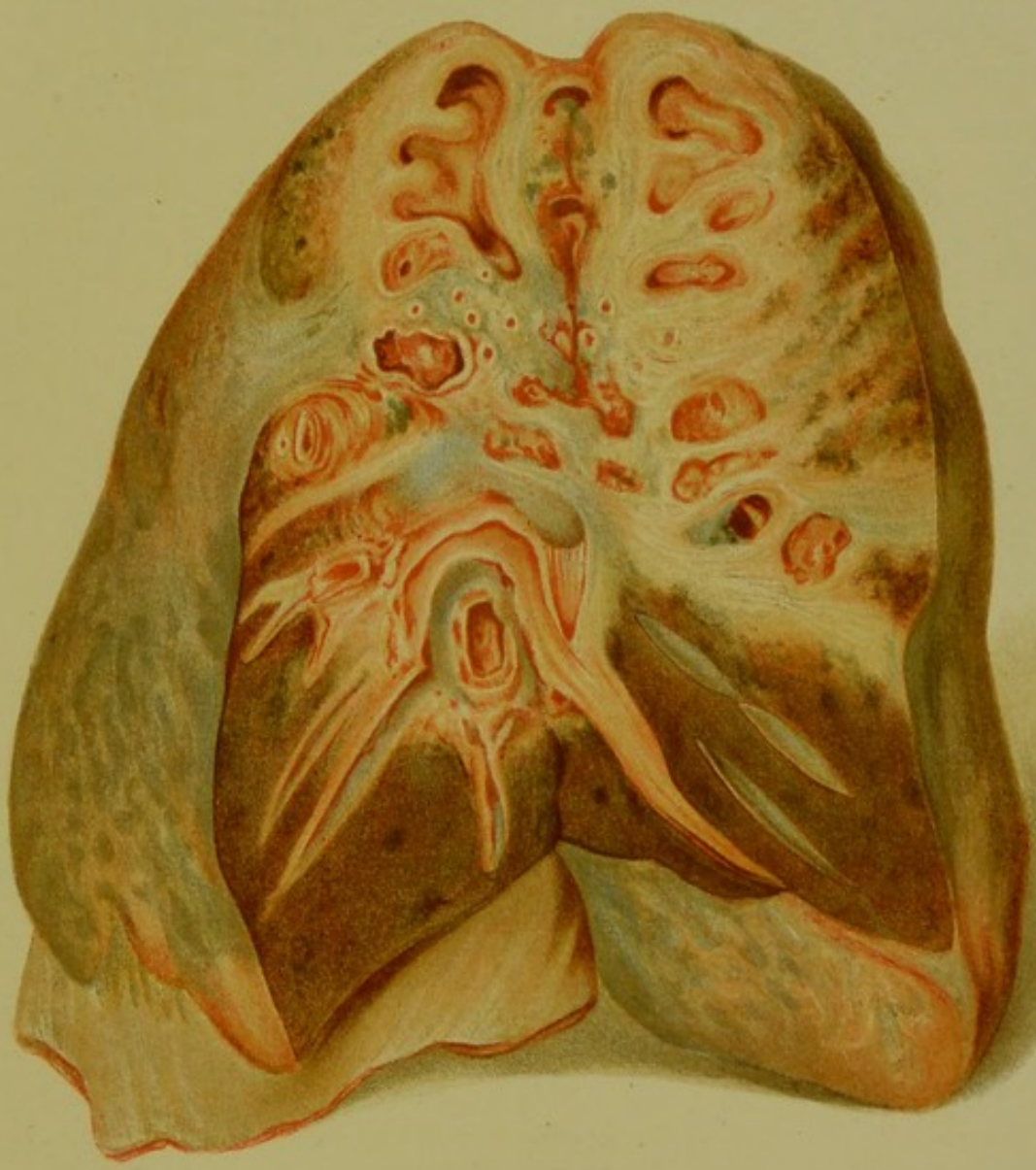
There had been varying cough from the age of one year, with distinct clubbing of fingers and cyanosis, both increasing from that period onwards. After an attack of measles the cough became for a time more severe. Sharp râles were heard over both backs, and a very fœtid smell of the breath was observed. Then the cough and râles diminished, but the cyanosis and clubbing increased, and the child's breath was shallow, and sometimes wheezy.

From the age of three months the child suffered from repeated convulsions, which affected the left side more than the right.

The intra-ocular growths above described had been observed with the ophthalmoscope during the first year of life, and they had slowly enlarged. They may possibly have been partly congenital.

The child had learnt to walk a little, but not to speak.

He died in a convulsion, his lung trouble not having shown any special aggravation during the last few weeks of his life.



necessary, and the exhibition of quinine and tincture of iron frequently of service. A septic form of pneumonia may arise, but no special treatment, beyond that already mentioned, is available. Sometimes it will be necessary in such cases to check profuse diarrhœa; and should pyæmia with secondary deposits supervene, they may need surgical interference.

5. **Recurrent Pleurisy.**—The "dry pleurisy," which is the form associated with, and giving rise to fibroid disease, is often most insidious, and generally apt to run a chronic course, and to constantly return. Beyond the general rules laid down above, in order to prevent fresh outburst from unnecessary exposure, there is little to be done. Counter-irritation proves of use in some cases, and it is important to obtain for the patient as much rest as possible during the attack, remembering that movement will in all probability prolong it, and, by causing irritation, produce a more copious exudation of organisable lymph. This rest may be obtained, to a certain extent, by strapping the side, but is much more effectually gained, by rest in a recumbent position, and counter-irritants may at the same time be employed.

CHAPTER VI.

ANALYSIS OF THE CASES OF PURE FIBROID DISEASE.

In this chapter we propose to give an analysis of every case of pure fibroid disease, that has come under our notice; and in order to investigate fully all the points in the disease, recourse has been had to the statistical method. The cases are presented to the reader in a tabular form, and such inferences as could justly be drawn from a statistical consideration of these Tables have been appended. It will be, perhaps, well to observe that we fully appreciate at its proper value, the amount of weight to be attached to statistics when dealing with a limited number of cases. Although this number (45) is large for the, comparatively, rare fibroid disease, yet we should not be much surprised, if we found another series of 45 cases, in some particulars at least, giving different average results. Radicke's paper on the "Arithmetic Mean in Medicine" has done much to strip limited statistics of their importance, and to show that evidence which rests on such a basis, is not altogether unassailable. Yet, notwithstanding all this, we feel sure that in some particulars, weight, great weight, must be attached to the considerations we shall presently set forth, since they are gathered from a critical inquiry into the cases as a whole. Another important point to bear in mind is that these cases are entirely drawn from hospital practice, and one of us, who has observed a large number of cases in his own private practice, would, basing his conclusions on cases drawn from people in the better walks of life, hold different views as to cause and duration.

For instance, with regard to the **Age** of the patients, he finds that in private practice, the subjects of the disease are, as a rule, far older than hospital patients.

Age.—Bearing these facts in mind through the following pages, we will put on paper the results of a statistical inquiry into these cases.

TABLES OF CASES OF PURE FIBROID DISEASE UNCOMPLICATED BY TUBERCLE.

No. of Case.	Initials.	Sex.	Age.	Duration in Years.	Side.	Family History.	Personal History.	Appearance of Chest.	Heart.	Signs and Symptoms.	Tubercle Bacilli.	Temp.	Urine.	Pulse.	Fingers.	Remarks.
1	C. S.	M	11	9½	L.	None of tubercle or fibrosis.	Measles at 15 months; cough ever since. Whooping-cough 6 months after measles.	Flattened throughout, motionless, contracted, a few white spots.	Displaced to left and upwards; beats seen in left axilla. No bruit.	Dyspnœa and cough, summer and winter, considerable expectoration. Dulness over whole side. Bronchial breathing. Pectoriloquy, and coarse râles, heard chiefly at the base.	0	Normal.	No albumen.	Quiet.	Not clubbed.	
2	C. C.	M	8	8	R.	Paternal aunt and grandfather died of phthisis. No fibrosis.	Whooping-cough at 7 weeks, measles at 4. Cough ever since the whooping-cough.	Flattening of right side. Slight movements. Right shoulder dropped.	Inside right nipple, chiefly at ensiform cartilage. No bruit.	Cough, dyspnœa. Scanty expectoration ever since the whooping-cough. Sweating one year. Impaired resonance, weak breath sounds, scanty crepitations, no creaking, no cavernous sounds. Signs chiefly basic.	0	Normal.	No albumen.	Quiet.	Slightly clubbed	
3	R. V.	M	52	49	R.	None of tubercle or fibrosis.	Measles and whooping-cough at 3; cough ever since then. Worked in a wool house. Small-pox 30 years ago. Alcohol at times to excess.	Right side contracted and motionless. Diminished expansion. No white spots.	Displaced to right in the epigastrium. No bruit.	Dyspnœa, cough, expectoration, streaks of hæmoptysis. No sweating. Has lost one stone in ten years. Resonance impaired over right lung, and dull at extreme base. Absent breath sounds, scanty crepitations. Tactile vocal fremitus, and vocal resonance increased in infra-clavicular region. Œdema of legs.	0	Normal.	Much albumen.	Slow.	Fingers and toes very bulbous.	

No. of Case.	Initials.	Sex.	Age.	Duration in Years.	Side.	Family History.	Personal History.	Appearance of Chest.	Heart.	Signs and Symptoms.	Tubercle Bacilli.	Temp.	Urine.	Pulse.	Fingers	Remarks.
4	L. M.	F	7	5	L.	None of tubercle or fibrosis.	Measles at 2 years; whooping-cough 6 weeks later, cough ever since.	Slight flattening and diminished expansion. No spots.	Two inches outside left nipple. No bruit.	Dyspnœa, paroxysmal cough ending in vomiting. Fœtid expectoration. No hæmoptysis. Slight wasting and sweating. Chest impaired all over; dull at base. Bronchial breathing, and pectoriloquy at base. Much coarse crepitation.	0	Normal.	No albumen.	Quiet.	Fingers bulbous.	? Syphilis.
5	A. G.	M	14	10	L.	None of tubercle or fibrosis.	Whooping-cough when 4 years old, followed by bronchitis and inflammation of lung. Measles soon after. Cough since then.	Flattened, contracted, diminished movement.	Two inches outside left nipple. No bruit.	Dyspnœa, cough, expectoration all through the year. Streaky hæmoptysis. Chest impaired all over, dull at base. Bronchial breathing and bronchophony in the posterior axillary region. Creaking sounds and crepitations.	0	Normal.	No albumen.	Quiet.	Fingers bulbous.	No reaction to tuberculin.
6	M. B.	F	11	7	R.	None of tubercle or fibrosis.	When 4 had bronchitis and inflammation of lung followed by whooping-cough.	Expansion equal on both sides. A few white spots.	Not displaced. No bruit.	Slight dyspnœa and cough with scanty expectoration. Impaired note all over back and front, no cavernous sounds anywhere, sharp crepitation and creakings at apex. Breath sounds absent at base.	0	Normal.	Albumen in small quantities.	Slow.	Fingers slightly bulbous.	

7	M. S.	F	19	15	L.	Mother's brother died of phthisis. No fibrosis.	Measles at 4 ; cough ever since, and latterly epistaxis.	Contracted, no movement. Lateral curvature.	Displaced upwards. No bruit.	Paroxysmal cough ending in vomiting, dyspnœa not excessive ; streaky hæmoptysis. Note against left apex (this probably due to curvature). crepitation all over, no bronchial breathing.	0	Normal.	Albumen in small quantities.	Quiet.	Fingers not clubbed.
8	M. B.	F	8	6	L.	None of tubercle or fibrosis.	Measles and whooping-cough at 2. Cough ever since.	Contracted, diminished movement. A few white spots.	Apex-beat displaced outwards. No bruit.	Slight dyspnœa, cough and expectoration. No hæmoptysis, much sweating. Note against whole of left chest, back and front. Bronchial breathing and bronchophony at angle of scapula, crepitations and creakings.	0	Normal.	No albumen.	Diminished	Fingers slightly clubbed.
9	L. H.	F	17	14	R.	None of tubercle or fibrosis.	Whooping-cough followed by bronchitis. Cough since then.	Flattening and contraction at right base; expansion deficient.	Heart in normal position. No bruit.	Dyspnœa, severe on exertion, cough summer and winter, of paroxysmal character in morning, often ending in vomiting. Pain in side. Dulness in right axillary region, crepitations coarse. Apex clear.	0	Normal.	No albumen.	Quiet.	Thumbs clubbed.
10	M. A.	F	9	9	L.	Father died of phthisis. Mother's family phthisical. No fibrosis.	Whooping-cough when 3 weeks old ; cough since then.	Flattening, deficient expansion over lower part of left lung. No white spots.	Displaced; at fourth interspace in anterior axillary border. No bruit.	Slight dyspnœa. Expectoration comes up in gushes, slight emaciation, no hæmoptysis. Patient is well nourished. Dulness lower two-thirds of left lung. Bronchial breathing, pectoriloquy, and crepitation, especially at inferior angle of scapula.	0	Normal.	No albumen.	Quiet.	Fingers clubbed.

No. of Case.	Initials.	Sex.	Age.	Duration in Years.	Side.	Family History.	Personal History.	Appearance of Chest.	Heart.	Signs and Symptoms.	Tubercle Bacilli.	Temp.	Urine.	Pulse.	Fingers.	Remarks.
11	A. E.	M	16	14	L.	Father died of a "broken blood-vessel." No fibrosis.	Has been ailing since 2 years old, then had measles, followed by "chest disease." Cough and expectoration since measles.	Flattening of left side, deficient expansion and movement. A few white spots.	Displaced fourth space, anterior axillary line. No bruit.	Dyspnœa on exertion. Cough and expectoration summer and winter, never quite abating. Has never had any hæmoptysis. Impaired note all over left side, dull at base. Loud bronchial breathing over limited area, at inferior angle of scapula, with bronchophony. A few crepitations.	0	Normal.	Albumen in small quantities.	Quiet.	No clubbing.	
12	W. P.	M	23	15	R.	None of tubercle or fibrosis.	Measles when 8 years old, since then liable to cough. Six years ago cough and expectoration became much worse.	Flattening and impairment of movement over the right side. No white spots.	Displaced. Apex-beat seen 1 inch outside right nipple in fifth interspace. No bruit.	Dyspnœa on exertion, Cough and expectoration not paroxysmal; always worse on exertion. Slight streaky hæmoptysis. No loss of flesh. No sweating. Epistaxis. Dulness more or less complete, and absolute as the base is reached. Bronchial breathing at lower part, pectoriloquy. Much moist crackling and dry creaking.	0	Normal.	No albumen.	Quiet.	No clubbing.	
13	E. M.	F	41	25	L.	None of tubercle or fibrosis.	Measles when young. At 16 in St. Mary's Hospital with "con-	Contracted, flattened, absence of movement. Slight expansion.	Sixth space, anterior axillary line. Presystolic	Since rheumatic attack breath has become shorter. Cough, expectoration now. Did have sweating. Slight loss of flesh. No hæmoptysis.	No expectoration.	Normal.	No albumen.	Quiet.	Clubbed.	

							sumption." At 25 had rheumatism; said to have coughed since the measles, on and off.		murmur and thrill.	Dull all over, especially at base. Very few moist sounds at all. Bronchial breath sounds and bronchophony at apex and inferior angle of scapula. Spleen and stomach drawn up.						
14	A. H.	F	14	13	L. & R.	None of tubercle or fibrosis.	Measles and whooping-cough when a few months old. Since then has coughed incessantly.	Both sides of chest contracted at bases, most left; both sides immovable at bases.	Not displaced. Still within nipple line. No bruit.	Dyspnœa, cough, and expectoration, paroxysms in morning. No loss of flesh, no hæmoptysis. Dulness at both bases, back and front; extending higher up on left side. Bronchial breathing and bronchophony at both bases. Copious metallic crepitation at both bases.	0	Normal.	No albumen.	Quiet.	Very clubbed.	
15	F. G.	F	26	23	L.	None of tubercle or fibrosis.	When young scarlet fever, measles and whooping-cough. At 3 began to cough. In hospital eleven times.	Left side flattened and motionless.	In situ. No murmur.	Dyspnœa slight. Cough and expectoration summer and winter, no great loss of flesh. Hæmoptysis 12 years ago. Note impaired over left side from apex to base. There can be heard cavernous breath sounds and voice sounds, attended with superficial crepitations. (Crepitations in right axilla.)	0	Normal.	A trace of albumen.	Quiet.	Slightly clubbed.	

No. of Case.	Initials.	Sex.	Age.	Duration in Years.	Side.	Family History.	Personal History.	Appearance of Chest.	Heart.	Signs and Symptoms.	Tubercle Bacilli.	Temp.	Urine.	Pulse.	Fingers.	Remarks.
16	W. R.	M	12	10	L. & R.	None of tubercle or fibrosis.	Measles and whooping-cough when 2 years old. Cough on and off ever since.	Flattening and contraction of both sides of the chest at base.	In situ. No bruit.	Cough, dyspnœa, expectoration, worse in winter, never entirely ceasing. Note impaired at left base. Breath sounds weak. At inferior angle of scapula bronchial breathing with bronchophony, loud creaking and crepitations. Some crepitations at *right base.*	0	Normal.	No albumen.	Quiet.	Clubbed.	
17	F. W.	M	8	3	L.	None of tubercle or fibrosis.	At 5 years of age had whooping-cough. Cough ever since.	Impairment of movement over left side. No white spots.	Displaced half inch outside nipple. No bruit.	Dyspnœa, cough. Expectoration and wasting set in after whooping-cough. No loss of flesh now, no hæmoptysis. Note impaired all over left side. Breath sounds attended with crepitations, especially at base.	0	Normal.	No albumen.	Quiet.	No clubbing.	
18	H. R.	M	14	13	L.	One sister died at 18 of phthisis. History of cough throughout family. Mother has had four miscarriages.	Whooping-cough when 6 weeks old. Cough ever since then.	Impaired movement over left side. No white spots.	Displaced; fourth space 1½ inches outside nipple. No bruit.	Dyspnœa, cough. Expectoration, no wasting, no hæmoptysis. Has sweated considerably. Dulness all over left side. Bronchial breathing at mid-scapular region, large coarse crepitations (deficient note at right base).	0	Not taken.	Not tested.	Quiet.	Slightly clubbed.	Fissures and scars at angles of mouth.

						tubercle. Father died of morbus cordis. Mother died of gout. One sister has morbus cordis. History of fibrosis.	Whooping-cough at 4 years old. Bad cough ever since. Pleurisy July, 1891.	Contraction; impairment of movement over left side.	Displaced; 3½ inches outside nipple No bruit.	Dyspnœa, cough, expectoration, no hæmoptysis, no sweating, no emaciation. Dulness from apex to base. No adventitious sounds. Bronchial breathing and bronchophony at inferior angle of scapula.	0	Normal.	No albumen.	Not observed	Not clubbed.	
20	M. M.	F	32	10	R.	None of tubercle or fibrosis.	Measles followed by trifling cough. Ten years ago caught cold, then cough. Expectoration very bad.	Contraction; deficient movement; no white spots.	Displaced; fifth interspace under right nipple. No bruit.	Dyspnœa. Cough summer and winter. Profuse expectoration, comes up in gushes, never fœtid; no wasting, no hæmoptysis. Note impaired all over, dull at base. Tactile vocal fremitus increased. At base, coarse crepitation, bubbling sounds, bronchial breathing and bronchophony. A few crepitations at left base.	0	Normal.	Not tested	Quiet.	Clubbed	
21	D. H.	M	22	14	L.	Father died of phthisis at 40. No history of fibrosis.	Whooping-cough when young, also measles; slight cough after that; fourteen years ago "congestion of lungs." Has had rheumatic fever.	Flattening, contraction, deficient movement; no white spots.	Displaced; apex 2½ inches outside nipple. No bruit.	Dyspnœa on exertion. Cough and expectoration summer and winter. No sweating; no emaciation. Two attacks of hæmoptysis. Epistaxis. At apex, note hyperresonant. Dull from mid-scapula to base. Tactile vocal fremitus increased. Bronchial breathing and pectoriloquy at base. Coarse creaking and crepitations. Stomach pulled up.	Five years ago 0, now 0	Normal.	5 yrs. ago none, now much albumen.	Quiet.	Very clubbed.	

No. of Case.	Initials.	Sex.	Age.	Duration in Years.	Side.	Family History.	Personal History.	Appearance of Chest.	Heart.	Signs and Symptoms.	Tubercle Bacilli.	Temp.	Urine.	Pulse.	Fingers.	Remarks.
22	A. D.	F	16	15	L.	None of tubercle or fibrosis.	Whooping-cough when a baby; since then cough has been present summer and winter.	Deficient expansion at base; no white spots.	More exposed than usual; apex-beat outside nipple. No bruit.	Cough, expectoration. No hæmoptysis. No wasting. Sweating and diarrhœa at times. Note against left lung. Crepitations and creaking. Weak breath sounds at base.	0	Normal.	No albumen.	Quiet.	Slightly clubbed.	
23	G. B.	M	14	9	L & R.	Father has nephritis. Two uncles died of bronchitis. Mother has bronchitis. No tubercle.	Slight cough at 6 months; when 5 years old had whooping-cough and measles, and then cough became excessive.	Contraction and diminished expansion; a few white spots. Harrison's sulcus.	Not much displaced. No bruit.	Dyspnœa, cough and expectoration, paroxysmal in morning. No diarrhœa, no hæmoptysis, no night sweats, no emaciation. Note impaired all over, and nearly dull at base. Loud coarse mucous râles all over. (A few crepitations at right base.)	0	Normal.	A fair cloud.	Quiet.	Slightly clubbed.	
24	I. W	F	8	4	L.	Three brothers have morbus cordis. Grandfather died of dropsy. No tubercle.	No measles or whooping-cough; 4 years ago sudden cough; blue in face; evident dyspnœa; cough ever since.	Contraction and diminished movement; no white spots.	Displaced outside nipple; no murmur.	Cough, dyspnœa, expectoration. Slight loss of flesh. Slight hæmoptysis. Night sweats considerable, no diarrhœa. Left lung impaired all over, dull at base; crepitations all over left lung, attended with loud bronchial breathing, and bronchophony at inferior angle of scapula.	0	Normal.	No albumen.	Quiet.	Clubbed.	

25	E. I.	F	22	6	L. & R.	Father died of morbus cordis. Mother died of apoplexy; none of tubercle.	Hacking cough some years; pneumonia 6 years ago; several like attacks since; never whooping-cough; no other illness at any time.	Much contraction; deficient movement and expansion of left side; no white spots.	Displaced; 1½ inches outside nipple; no murmur.	Excessive dyspnœa. Expectoration in the morning on waking. No loss of flesh, no sweating; pain in left side. Hæmoptysis. Dull note from apex to base. Breath sounds cavernous, attended with loud, coarse gurgling sounds and pectoriloquy. (Right lung at base, impairment of note, and crepitations.)	0	Normal.	$\frac{1}{5\frac{1}{0}}$th albumen.	Quiet.	Clubbed.		
26	E. S.	F	40	10	R	None of tubercle; mother's family morbus cordis.	Small-pox at 5; scarlet fever in childhood; measles when 24; ten years ago inflammation of right lung which never resolved; cough since then.	Contraction, diminution of movement, deficient expansion; no white spots other than small-pox marks.	Displaced; apex-beat 1 inch to right of sternum in fifth space; no murmur.	Cough, expectoration, and dyspnœa. No loss of flesh. Hæmoptysis considerable. Pain in right side. Diarrhœa at times. Impaired note all over, dull at base. Bronchial breathing with coarse crepitations, and bronchophony. No evidence of distinct cavity.	0	Normal.	No albumen.	Quiet.	Clubbed.	Heart was not displaced 3 years ago.	
27	M. H.	M	24	19	L.	None of tubercle or fibrosis.	Measles when a child; followed by "chest complaint"; cough ever since; alcoholic.	Contraction, impairment of movement, deficient expansion.	Displaced; 1½ inches outside nipple; no murmur.	Cough summer and winter. Expectoration coming up in gushes in morning, never fœtid. Well until 10 months ago. Profuse sweats, loss of flesh and hæmoptysis. Dull note all over, absence of breath sounds and vesicular sounds. No crepitations at all. Thickened pleura.	0	Subfebrile.	No albumen.	Quiet.	Very clubbed, also toes.	? Tubercular.	

No. of Case.	Initials.	Sex.	Age.	Duration in Years.	Side.	Family History.	Personal History.	Appearance of Chest.	Heart.	Signs and Symptoms.	Tubercle Bacilli.	Temp.	Urine.	Pulse.	Fingers.	Remarks.
28	R. S.	M	14	12	L.	Father died of phthisis ; no fibrosis.	Whooping-cough and measles at 3 weeks old ; the cough never has left him.	Deficient movement and expansion over left side.	*Not* displaced ; no murmur.	Cough, dyspnœa, scanty expectoration, no great loss of flesh. No hæmoptysis. No night sweats. Note against the left side, no real dulness anywhere. Coarse râles all over left side, especially at left base.	0	Normal.	No albumen.	Quiet.	Not clubbed.	
29	E. M.	F	14	12	R.	Mother's father died of phthisis ; none of fibrosis.	Whooping-cough when 1½ years ; cough ever since.	Contraction, deficient movement and expansion ; no white spots.	Displaced; apex-beat in fourth space under *right* nipple. No bruit.	Paroxysmal cough in morning on waking. Dyspnœa. No hæmoptysis. Trifling wasting. Pain in right side, no diarrhœa. Drooping of right shoulder, dulness from apex to base. Tactile vocal fremitus increased. Bronchial breathing, especially at mid-scapular region. Loud coarse gurgling crepitations.	0	Normal	No albumen.	Quiet.	Clubbed slightly.	Turgid countenance ; undergrown.
30	M. S.	F	28	20	L.	Father's sister died of phthisis ; none of fibrosis.	Measles and whooping-cough at 4 ; cough ever since, summer and winter.	Flattening, contraction, diminution of movement ; white spots in abundance.	Displaced; apex-beat in mid-axilla ; pulled upwards. No bruit.	Paroxysmal cough on waking. Expectoration comes in gushes. No loss of flesh, no diarrhœa. Hæmoptysis occasionally. Vocal resonance increased. Note dull from apex to base. Loud bronchial breathing and pectoriloquy in axilla, and at inferior angle of scapula. Coarse gurgling crepitations heard all over lung.	0	Normal.	No albumen.	Quiet.	Clubbed.	Looks the picture of health ; well-grown.

31	H. C.	F	27	11	R.	Father died of dropsy and congestion of lungs; None of tubercle.	Whooping-cough and measles at 7; cough since; well in health until 11 years ago; then she had bronchitis and inflammation of lungs.	Contraction; diminution of movement; lateral curvature; no white spots.	Impulse seen under right nipple in fourth space. No bruit.	Cough, dyspnœa, expectoration. Hæmoptysis 9 years ago. Sweating and emaciation recently. Vocal resonance increased. Note impaired all over, dull at base. Breath sounds cavernous and amphoric; pectoriloquy. Gurgling and bubbling râles at base and inferior angle of scapula.	0	Normal	No albumen.	64	Not clubbed.	
32	M. C.	F	11	6	R.	Father died of phthisis; none of fibrosis.	Cough since 5 years old, when she had some chest complaint; ? whooping-cough.	Impairment of movement over the right side; not much contraction.	Not displaced. No bruit.	Cough, dyspnœa, expectoration. Never hæmoptysis. No wasting. Slight impairment of note over right side. All over there are large, coarse, gurgling râles, especially at base.	0	Normal.	No albumen.	Quiet.	Not clubbed.	Not seen for 2½ years.
33	I. B.	M	22	18	L.	None of tubercle or fibrosis.	Been ailing since whooping-cough at 4; much worse after inflammation of lungs at 11.	Left side contracted, motionless; white spots.	1 inch outside nipple. No bruit.	Dyspnœa on exertion. Cough. Fœtid expectoration. Slight hæmoptysis. No wasting, no sweating. Dull from apex to base. Tactile vocal fremitus increased. Bronchial breathing, and bronchophony, attended with gurgling râles, especially at inferior angle of scapula.	0	Normal.	No albumen.	Quiet.	Clubbed.	Died. Post-mortem.

No. of Case.	Initials.	Sex.	Age.	Duration in Years.	Side.	Family History.	Personal History.	Appearance of Chest.	Heart.	Signs and Symptoms.	Tubercle Bacilli.	Temp.	Urine.	Pulse.	Fingers.	Remarks.
34	I. B.	M	26	6	L.	None of tubercle or fibrosis.	Winter cough many years; no definite illness.	Well-formed except at left base, where there is flattening.	Not displaced. No bruit.	Cough, expectoration coming up in gushes every morning on waking. Used to sweat profusely, and emaciated much; now not so. No hæmoptysis. Impairment of note at left base. Weak breathing, attended with loud bubbling râles, no bronchial breathing or bronchophony.	0	Normal.	No albumen.	Quiet.	Not clubbed.	A very slight cavity.
35	H. C.	M	65	6	R.	Mother died of dropsy; nothing else known.	No definite illness; winter cough for 6 years; much worse 16 months ago.	Right side contracted; impairment of movement all over right side.	Displaced to the right; impulse in epigastric region. No bruit.	Cough, expectoration, wasting. Streaky hæmoptysis. Great weakness, no diarrhœa. Dulness complete from apex to base, a few scattered crepitations all over. At mid-scapular region there are cavernous breath sounds. Left lung free.	0	Normal for 24 weeks.	No albumen.	Quiet.	Not clubbed.	? Fibroid disease, or new growth.
36	I. G.	F	23	16	L.	None of tubercle or fibrosis.	Measles and whooping-cough at 5; cough summer and winter for 16 years.	Contraction and impairment of movement of left side; no white spots.	One inch outside nipple line. No bruit.	Cough, dyspnœa, wasting; no hæmoptysis or sweating. Dulness all over left side, more at base. Bronchial breathing, loud coarse crepitation, and evidence of a vomica at inferior angle of scapula.	0	Normal.	No albumen.	Quiet.	Slightly clubbed.	Looks the picture of health.

37	R. S.	F	12	3	L.	None of tubercle or fibrosis.	Scarlet fever 3 years ago; measles when very young; cough after measles summer and winter; since scarlet fever cough has become worse.	No contraction; slight impairment of movement at left base. Many white spots.	One-third inch inside nipple; no murmur.	Cough, expectoration, nothing else to complain of. Impairment of note over left base. Signs of cavity there, crepitations.	0	Normal.	No albumen.	Quiet.	Congested ends.	Quite well.
38	B. S.	F	11	10	L.	Mother has chronic bronchitis and dropsy; fibrosis in family.	Bronchitis and inflammation at 1 year; cough and expectoration since then.	Left chest flattened and contracted; impairment of movement.	Displaced; half inch outside nipple. No bruit.	Cough, expectoration, dyspnœa. Streaky hæmoptysis. No night sweats. No wasting until recently. Expectoration is attended with vomiting. Evidence of a cavity at the apex in front. Coarse crepitations behind, some in front at inferior angle of scapula.	0	Normal.	No albumen.	Quiet.	Slightly clubbed.	
39	E. G.	F	10	2	L.	One brother died of "consumptive bowels"; no history of fibrosis.	Measles and whooping-cough 2 years ago; cough more or less ever since.	Chest flattened on left side at base.	Displaced; two inches outside nipple.	Cough, expectoration, dyspnœa. No hæmoptysis. Impaired note at left base and in axilla; coarse crackling crepitations and cavernous breath sounds at inferior angle of scapula.	0	Normal.	No albumen.	Quiet.	Not clubbed.	

No. of Case.	Initials.	Sex.	Age.	Duration in Years.	Side.	Family History.	Personal History.	Appearance of Chest.	Heart.	Signs and Symptoms.	Tubercle Bacilli.	Temp.	Urine.	Pulse.	Fingers.	Remarks.
40	E. H.	F	7	2½	R.	Father and sister died of phthisis.	Measles 2½ years ago, followed by "congestion of lungs"; cough ever since.	Flattened and contracted on the right side; no white spots.	Displaced to right; no murmur.	Cough, expectoration, dyspnœa. Slight hæmoptysis. No emaciation or night sweats. Impairment of note all over lung. Dull at base, evidence of cavities. Crepitations, creaking and gurgling.	0	Normal.	0	Quiet.	Clubbed.	
41	A. K.	M	2½	2	R.	None of tubercle or fibrosis.	When 7 months old had bronchitis. Cough, wheezing, and "rattling in chest" ever since.	No flattening on either side.	Displaced sixth space nipple line *right* side. No bruit.	Cough, not much dyspnœa or expectoration. Slight loss of flesh. Cough bad in morning, with gushes of phlegm. No hæmoptysis, no diarrhœa. Right lung; note impaired from the second rib; very dull at base. Bronchial breath sounds. Bronchophony, with hollow coarse râles. Left lung free.	0	99°–101° while in hospital.	No albumen.	144	Much clubbed.	Post-mortem.
42	T. M.	M	14	7	R.	None of fibrosis; but father has phthisis.	No previous illness to account for present condition; has had cough and expectoration for 7 years.	Right side considerably flattened; impaired movement; deficient expansion.	Displaced to right. No bruit.	Cough, expectoration, and dyspnœa summer and winter. Comes on especially in the morning. Right side of chest, especially lower two-thirds, note impaired. Breath sounds bronchial (especially at the angle of scapula), and attended with creaking and crepitations.	0	Normal.	No albumen.	Quiet.	Slightly bulbous.	

43	M.A.F.	F	9	3	L.	None of fibrosis. Uncles died of phthisis.	Cough, slight since birth, but became much worse after pleurisy, 3 years ago.	Left side flattened, more at apex; deficient movement and expansion.	Not displaced. No bruit.	Cough, expectoration, and dyspnœa since birth (more since an attack of pleurisy), coming on in morning, attended with vomiting. Slight hæmoptysis. No emaciation or night sweats. Slight impairment, crepitations creaking.	Sputum unobtainable.	Normal.	No albumen.	Quiet.	Clubbed.	
44	S. S.	F	42	12	R.	Father and one sister died of phthisis; none of fibrosis.	Cough, came on gradually without any illness to account for it.	Right side flattened all over; deficient movement and expansion.	Displaced to the right; impulse in epigastrium. No bruit.	Cough, expectoration, dyspnœa. Slight hæmoptysis. No wasting, but profuse sweating. Impaired resonance from apex to base, dull at base; cavernous sounds at apex, back and front, and at base behind; scanty crepitations and much creaking.	0	Normal.	No albumen.	Quiet.	Slight clubbing.	
45	E. K.	F	47	20	L. & R.	None of fibrosis or phthisis.	Cough on and off for the last 20 years, worse in winter; nothing to account for it.	Flattening at both apices; deficient movement there.	Not displaced. No bruit.	Cough, expectoration, and dyspnœa. Trifling emaciation and night sweats. No history of hæmoptysis. Impairment of note at both apices. Crackling and creaking and crepitations. Bronchial breathing and bronchophony.	0	Not raised.	A trace.	Quiet.	No clubbing.	

The total number of cases collected, in which there could be found no evidence of tubercle, was 45. Of these, 18 were males and 27 females. The average age of the 45 cases was found to be about 20 years. On comparing the average age of the males with that of the females, no notable difference could be found; the average age of the males working out to 20·1 years, that of the females to 19·9 years. To push these conclusions further: the age of oldest patient coming under notice was 65 years, of the youngest 2½ years. In the following Table the number of cases met with at different periods of life can be seen at a glance:

Age of Patients.

Between the age of 1 and 5 years we observed 1 case

,,	,,	5	,, 10	,,	,,	8	cases
,,	,,	10	, 15	,,	,,	14	,,
,,	,,	15	,, 20	,,	,,	4	,,
,,	,,	20	,, 25	,,	,,	6	,,
,,	,,	25	,, 30	,,	,,	5	,,
,,	,,	30	,, 35	,,	,,	1	,,
,,	,,	35	,, 40	,,	,,	1	,,
,,	,,	40	,, 45	,,	,,	2	,,
,,	,,	45	,, 50	,,	,,	1	,,
,,	,,	50	,, 55	,,	,,	1	,,
,,	,,	55	,, 65	,,	,,	1	,,

These columns, when put in the form of a chart, show still more readily at what periods of life these cases are most commonly met with. From a study of this Table, and the accompanying chart, it will be seen that, out of the 45 cases, no less than 38 were observed between the ages of 1 and 30 years, and only 7 between the ages of 30 and 65. Of these 38 cases, 22 occurred between the ages of 5 and 15. These facts warrant the conclusion that the disease is rarely met with after the age of 30, as far as these statistics extend.

On comparing the periods of life of the male cases with those of the female, one meets with no appreciable difference, as the following Table will show.

Thus:

Of males between 1 and 30 years 16 cases were observed

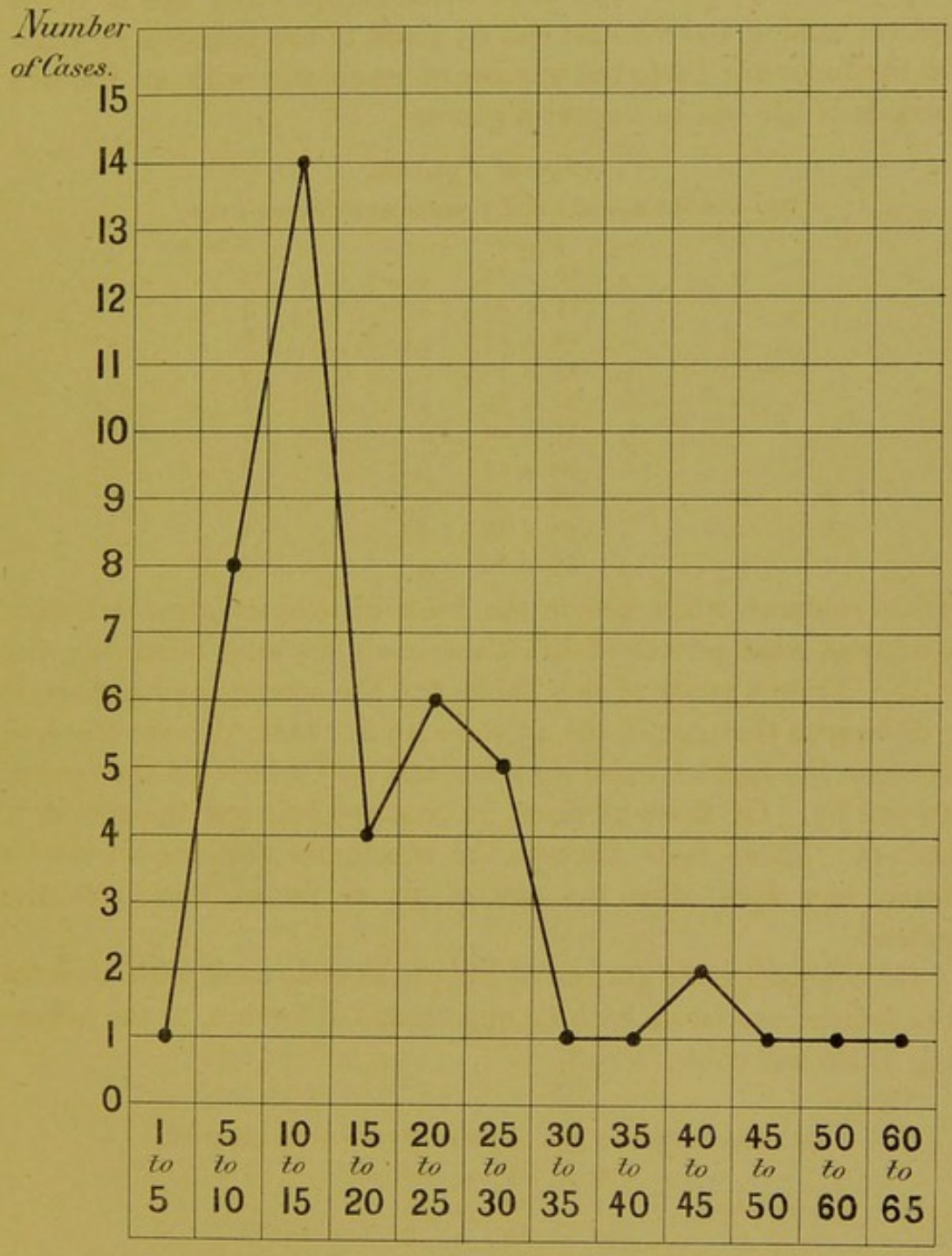
,,	,,	30	,, 65	,,	2	,,	,,
,, females	,,	1	,, 30	,,	22	,,	,,
,,	,,	,,	30 ,, 65	,,	5	,,	,,

Or again:

,, males	,,	5	,, 15	,,	9	,,	,,
,, females	,,	5	,, 15	,,	13	,,	,,

CHART.

Showing the Number of Cases met with at different periods of Life, from 1 to 65 Years.

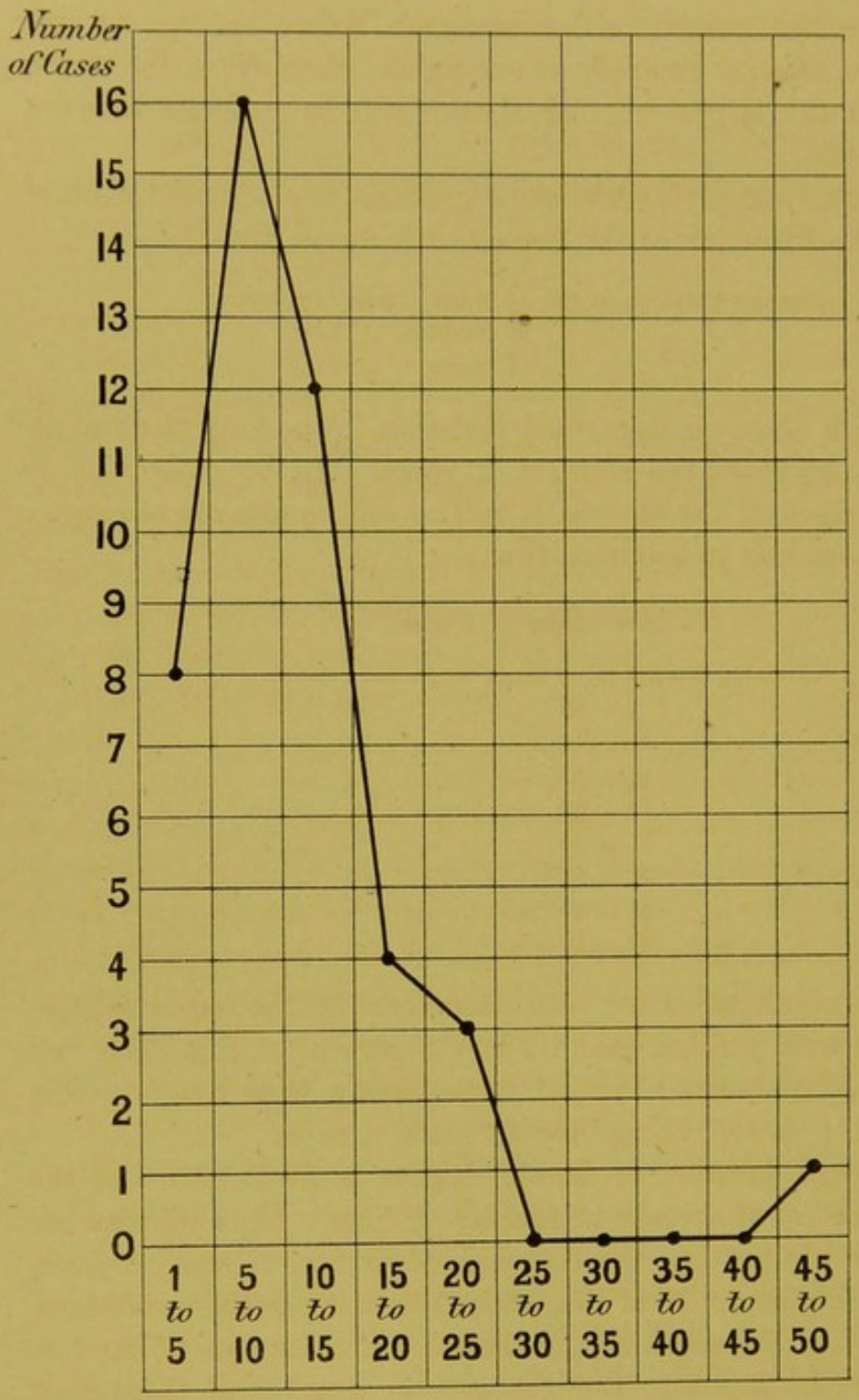


Ages of the Cases.

Danielsson & Co, lith.

CHART.

Showing the Duration in Years of the Cases met with.



Duration of Cases in Years.

Danielsson & Co. lith.

Reviewing, now, the conclusions brought out by this study of the ages at which fibroid disease is most common, and always remembering that the cases are drawn from a class of the community, in which the struggle for existence is keen, we are able to assume that the disease is not prone to develop much after 30 years; that the greater number either succumb before the age of 30, or at all events cease to apply at our public institutions for relief. Lastly, that the largest number of cases will be met with between 5 and 15 years.

Duration.—Applying the same methods to elucidate the chief points in the **Duration** of the disease, it is found that the

Average duration of the	45	cases	was	11·8 years
„	„	27 females	„	11·2 „
„	„	18 males	„	12·4 „

figures which show no important variation. The longest duration noticed was 49 years, the shortest 2 years. To be clearer as to the exact length of the disease, it will be well to arrange the cases, and their durations in columns, thus:

Duration of Cases.

Lasting from	1	to	5 years	we found	8 cases
„	5	„	10 „	„	16 „
„	10	„	15 „	„	12 „
„	15	„	20 „	„	5 „
„	20	„	25 „	„	3 „
„	25	„	30 „	„	0 „
„	30	„	35 „	„	0 „
„	35	„	40 „	„	0 „
„	40	„	50 „	„	1 „

The same result, when put into chart form, on the opposite page, can be seen with greater ease. From a perusal of this Table and chart, it will be observed that, of those lasting from 1 to 20 years, we found 41; those, lasting over 20 years, 4 cases.

This average duration of fibroid disease is far in excess of the average duration of chronic tubercular phthisis. No authority has yet succeeded in putting the duration of phthisis beyond 8 years, even in the higher ranks of life, under the best possible conditions, and measuring the length of the disease from the date of onset to the death of the patient. But the greater number of these cases before us are still living, and in the enjoyment of excellent health; and although they are hospital cases, and have to struggle amid

harassing surroundings, yet the majority of them have a fair prospect of life. Could we but follow all these cases to their termination, there can be little doubt but that the average duration would be far longer than 11·8 years.

On comparing the age with the duration, we find no more than one would expect—viz., the longer the duration of the disease, the greater the age. The figures are as follows:

Comparison of Age and Duration.

Of the 8 cases lasting from	1	to	5	years	the average age was	8	years		
„ 16	„	„	5	„ 10	„	„	„	19·8	„
„ 12	„	„	10	„ 15	„	„	„	19·8	„
„ 5	„	„	15	„ 20	„	„	„	28·8	„
„ 3	„	„	20	„ 25	„	„	„	31·6	„
„ 1	„	„	40	„ 50	„	„	„	52	„

These results bring us again to the fact, which has been already elucidated, that it is in the earlier periods of life that we must look for the beginning of the disease. A glance at the two last columns will show that, in the majority of cases, the date of onset of the disease was not after the age of 15.

Points of Interest in the Tables.—Several points in addition should be noted:

1. **The Side Affected:**

In 25 cases the left side alone was found diseased
„ 15 „ „ right „ „ „
„ 5 „ both sides were affected.

In each of the last five cases, however, the left side was in a more advanced condition than the right.

An attempt was made to ascertain whether disease of left side, offered a longer prospect of life, than that of the right, or *vice versâ*. As a result of these investigations, we found that the average duration of both the right and left-sided cases, was about equal to the average duration of all cases—viz., 11·8 years. But the average age was found to differ considerably; that of the right-sided cases being 24·4 years, that of the left 17 years. The average duration of those with both sides affected was 11·6 years, the average age 21·8 years. From a consideration of these statistics, it will be seen that the left side is most frequently affected. This statement is in agreement with many authors, among whom may be mentioned Sutton, Wilson Fox, and Sir Andrew Clark.

2. We observe, further, that these cases of double disease are of

as long duration as those of single disease. One might with propriety argue that, cases in which there was a double affection, would be of longer duration than others, since the affection, being primarily confined to one lung, would require a considerable period to elapse before the other lung became implicated. On the other hand, on the supposition that both lungs became simultaneously affected, one might infer that patients would succumb much more readily to the disease, and that the duration of the complaint would be thereby much shorter. But these statistics, scanty although they be, lend no support to the belief that double disease is more fatal than a one-sided affection.

3. Whilst the duration of both left and right disease was about 11·5 years, the average age of those with right disease was 24·4 years, of those with left, 17 years; so that the disease apparently comes on later in life in right-sided affections. This last result might be accidental, or due to the fact of a few isolated cases of old people, who happened to have right-sided mischief, upsetting the average in a comparatively small series. However that may be, this is certain that, in several of our right-sided cases, the disease originated in an attack of acute croupous pneumonia, coming on late in life, and not in broncho-pneumonia, following whooping-cough as is so often the case. This would offer a satisfactory explanation of the question.

4. **The Heart.**—The condition of the heart, as to its position, action, and size, was investigated with the utmost care in all cases. Of all the phenomena connected with the heart, displacement of the organ was the most constant. This we found present, in greater or less degree, in 34 out of the 45 cases. On investigating the 11 cases in which there was no displacement of the heart, the following state of things was found:

(*a*) In 4 cases there was right-sided disease in addition to the left, and one of these was a case of double apical disease.

(*b*) In 5 cases the disease was slight and of limited and trifling extent, being confined, for the most part, to the base of one lung.

(*c*) In 2 cases the disease was extensive, involving the whole of one lung.

Now 9 of these 11 cases furnish the cause of the absence of cardiac displacement. In 4 cases there was clearly marked disease in both lungs, and the extent of the disease was not much

greater in one lung than in the other. May not, then, the absence of displacement of the heart, in these 4 cases, be explained by supposing that the heart is retained in its position, by the opposing contracting forces, exerted upon it, by both lungs—that is, when both lungs have about an equal amount of disease? Of course, it cannot be denied that, when the disease in one lung is far in excess of the other, the heart, in spite of the traction exerted upon it, must still be drawn to the more diseased side. This last remark might afford an explanation of the reason why, in the remaining case of double disease, there was displacement of the organ. In this case the left lung was advanced in disease out of all proportion to the right.

The 5 cases, in which the disease was of limited extent, speak for themselves. So, in only 2 cases was there considerable one-sided disease and no displacement of the heart. In these cases, the heart may possibly have become fixed by adhesions, and so incapable of following the retracted lung.

The long duration of the case had apparently no effect upon the displacement.

The degree of displacement was subject to wide variation, from a half to 3 or 4 inches. When the displacement was to the left, the apex-beat could be defined with tolerable certainty. In one case it could be seen and felt at the edge of the posterior axillary fold. When dragged to the left, the heart is, as a rule, elevated in addition to being displaced outwards. In right-sided disease, the heart's impulse was found in all positions from the epigastrium to half an inch outside the right nipple line, in the fourth or fifth space. It was frequently quite impossible to say whether what one felt was the apex-beat, the ventricle, or the auricle. It is difficult to believe that the heart can bear, without impairment of its functions, such an amount of torsion as would transfer its apex from the fifth space on the left side, to a point on the right side, 8 or 9 inches away. Far more probable is it to assume that, the mediastinum and heart as a whole, are drawn over to the affected side. Indeed, *post-mortem* experience bears this out. In a case, recorded by Dr. Moxon in the *Transactions* of the Pathological Society, the apex-beat was found to be at the ensiform cartilage, while the ventricles and auricles were much further to the right.

In most of our cases, we were of opinion that it was the upper

part of the ventricle which we saw and felt beating, and not the apex.

The contractions of the heart were regular, strong, and measured.

In spite of most careful observations we were unable to detect cardiac murmurs, except in two instances, in both of which there was a clear history of rheumatic fever to account for them. Frequently, the pulmonary second sound was unduly loud and accentuated, but rarely attended with a bruit.

5. **The Pulse**, in all the cases coming under our notice, was of a uniformly quiet character, the beats seldom reaching 80 per minute; indeed, the average would not be more than about 70. The characters of the pulse were marked, and presented features of contrast to the pulse of a patient with tuberculosis. The beat was regular and full, measured or infrequent, and slow, using these terms in their strict sense. In some cases there was a certain amount of tension to be observed, and the arteries seemed to be somewhat rigid. The temperature, in all but two instances, was always normal. Sometimes we were able to observe the morning and evening variations, for a period extending over 6 or 12 weeks. In one case it was sub-febrile, and in one case slightly raised.

6. **Tubercle Bacilli.**—All cases were subjected to a careful examination for the bacillus; many of the cases we tested for the microbe 10 or 20 times, others not so often; but the result, in each case was precisely the same, absolutely negative; whereas, examination of expectoration from cases of chronic tubercular phthisis always, sooner or later, revealed the organism. The method we employed was that of Ziehl-Neelsen. In only 2 cases were we unable, on account of the absence of expectoration, to apply this test.

Family History.—1. **Tubercular History.**—In the 45 cases collected, a history of phthisis in the family was obtained in 13, whilst in the other 32, no such history was obtainable. In the first place, it must be said that this phthisical taint is not more frequent, than is found on the average in healthy individuals. On comparing the age and duration of the disease, in those cases, where a tubercular history was obtained, with the age and duration in those where there was no such history, no appreciable difference was noticed.

Thus, the average age of those with tubercular antecedents was 14 years, whilst the duration of the disease in them, was 10·3 years. The average age of those free from such taint, was 22·4 years, whilst in them, the disease had lasted 12·3 years.

2. **Fibroid History.**—In estimating this, questions were asked as to the presence in the family, of a history of heart disease, dropsy, nephritis, gout, rheumatism, or apoplexy. Such a history was obtained in 8 out of the 45 cases. The average age of these 8 cases was 27 years, whilst the duration of their lung trouble was 10·75 years, as against an average age of 18·6 years, and a duration of 12 years, for those without any such history being traceable.

Every case was carefully examined in order to obtain a history of fibroid diathesis, but without much success, as may be seen from the above figures, which speak for themselves. In many cases the information which should have been furnished by the patient or the friends, was very defective; and we are afraid that not much reliance can be placed upon the figures, and inductions, with reference to this point; they are, indeed, far too meagre and scanty. We must draw attention to one point more in the consideration of this subject, and that is, that although carefully inquired for, there was a total absence of any history of intemperance in the immediate antecedents of the patients.

3. **Syphilitic History.**—In two cases there was a history pointing to this as an inherited disease, and the patients, who were young (14 and 7 years respectively), showed by the teeth, and scars at the corners of the mouth, or by depressed nasal bridge, and history in infancy, that they were subject to this disease. In as far as we could discover, this taint did not affect in any way the nature, course, or duration of the disease in them; and, moreover, they both had had measles and whooping-cough in early life, from which attack the onset of the lung trouble dated.

Before discussing the signs and symptoms, a few points may be worthy of notice.

Assignable Cause.—Of the 45 cases, 33 of them attributed their disease to an attack of either measles or whooping-cough, and in many cases, both, occurring in early life; which attacks had been generally followed by broncho-pneumonia, bronchitis, or, at any rate, by some chest affection, the exact nature of which it was

difficult to ascertain. Of the remaining 12 cases the cause assigned was as follows:

A sudden attack of suffocative dyspnœa while at play, and cough ever since	1 case
Illness since a presumably *acute pneumonia*	2 cases
Measles followed by trifling cough, *pneumonia* 20 years later	1 case
Whooping-cough and measles followed by trifling cough at 7 years, *pneumonia* at 16, from which illness is dated	1 „
"*Chest trouble*" following *scarlet fever*	1 „
Pleurisy (Case 43)	1 „
No definite illness, but *winter cough* for many years	4 cases
Nothing to account for the illness	1 case
Total	12 cases

It will be seen that of these 12 cases, 4 were caused by "pneumonia," in 2 of which, there was a previous history of trifling cough after measles and whooping-cough.

In only one instance, could we satisfy ourselves that the disease had originated from a previous attack of pleurisy.

It is also worthy of note that those cases arising after a "pneumonia" or long-continued winter cough, began later in life than the others.

"White Spots" in the Skin.—In 6 cases we observed white spots of fibroid material in the skin of the chest, arms, or face. We found this so-called "sign" very inconstant, neither did we find any hypertrophy, undue contraction, or other morbid change, affecting scars, the result of accident. And we thought that these "white spots," were no commoner in our cases than in the most healthy individuals.

Abuse of Alcohol.—In only 2 cases could a history of intemperance be obtained. Indeed, the number of children and women formed such a large proportion of the total, that one would hardly have expected it to have been otherwise.

Albuminuria.—Great attention was paid to this sign, in all cases the urine being carefully tested.

Albumen was found in the urine of 9 cases out of the 45.

In 2 cases it was in	*large* amount.
„ 3 „ „	slight, but *appreciable.*
„ 4 „ „	only found in *faint traces.*

With regard to the duration of those cases in which albuminuria was present, the average was 17 years approximately, and this is some years in excess of the average duration of all cases, which

was 12 years, the duration in each individual case being 14, 9, 6, 14, 23, 49, 7, 15, and 20. It is here obvious that, in so small a number of cases, the duration of 49 years must upset the balance of calculation. Nevertheless, it does seem that the longer the duration of the disease the more prone is it to give rise to albuminuria, as may be seen in Case 21 in the Tables. In this case, when first seen, there was no albumen in the urine, the disease having lasted then for 9 years; but when the same case came under observation again, 5 years later, the urine was found loaded with albumen, the disease having then lasted 14 years. The longer the duration of the disease, other things being equal, the more lung-tissue tends to be affected; consequently the greater the congestion of the kidney from back pressure, and so the greater liability to the supervention of albuminuria. This is further borne out by the fact that albumen was present in 3 out of the 4 double cases. But in none of the cases of albuminuria could there be found any other evidences of constitutional fibroid degeneration.

Clubbing of Fingers.—This we found a fairly constant sign, being absent in only 13 out of 45 cases. The duration of the disease in those cases with "clubbing," was about 13 years, just over the average, whilst that of the cases which did not present this sign, was 11 years.

Symptoms.—The three cardinal symptoms, which were always present, were cough, dyspnœa, and expectoration. Very frequently, too, these were the only symptoms complained of, the common statement of the patient being that, but for the cough, shortness of breath and expectoration, he or she would be in perfect health. To take these and other symptoms severally:

1. **The Cough** was often found to be paroxysmal, chiefly on waking in the mornings, and at such times, was attended with gushes of expectoration, which were frequently so violent and prolonged that vomiting resulted. The cough and expectoration were generally observed to persist through summer, and, though often worse in winter, were, nevertheless, present at all seasons of the year.

2. **The Dyspnœa** was always excessive. This was noticed especially on the slightest exertion, in some cases; but in nearly every case, even when at rest, the breathing might be seen to be embarrassed, frequent, and shallow.

3. **The Expectoration** was in most cases profuse and lumpy,

though in a few, it was scanty and frothy. In two cases it was noted to be fœtid, and one of these died shortly after it had become so.

4. **Hæmoptysis** was not altogether absent in these cases. It was found to have occurred in considerable amounts in 7 cases, and to have streaked the expectoration in 10 others. It is a question how much importance is to be attached to these 10 cases, since most people, suffering from long-continued cough, or almost any chest disease, have their expectoration streaked with blood at some time or other. From the consideration of the cases presenting this sign it does not appear that it adds, in any way, to their gravity.

5. **Sweating** was found to have existed, at some time or other, in the disease in 10 cases only; in 6 cases it was profuse, whilst in the other 4 it was slight and insignificant in amount. It often had occurred years before, early in the disease, and in no case could we satisfy ourselves that it was the genuine sweat of pulmonary tuberculosis.

6. **Wasting.**—Several cases had emaciated slightly, at some period, during the existence of their chest trouble, and had again regained flesh and strength. This sign was found to be present in 10 cases, in 3 of which it was considerable, and in the remainder, only trifling in amount. The majority of the cases, however, presented the picture of health.

7. **Diarrhœa** was found in 2 cases only.

Physical Signs.—Each case presented its own peculiarities in this respect, and only a summary can be given of those usually present. All would not be present in every case, or perhaps, if present, would not be well-marked; but they were found, variously combined, in the different cases.

1. **Dulness** was almost always present, more or less marked, over part or the whole of the affected lung, being nearly always most complete towards the base, and sometimes being limited to that region. In only a few isolated instances, was it most marked at the apex.

2. **Contraction.**—This was, in nearly every case, a marked feature, accompanied by an equal impairment of movement.

3. **Vocal Vibration** to the hand and ear varied, being sometimes markedly increased, and at others diminished or wholly wanting; according as there was bronchial dilatation or cavity in the first

instance, or a considerably thickened pleura without such dilatation or cavity, in the last case.

4. **Bronchial Breath Sounds** were well heard, as a rule, at some spot of the affected lung. They were attended with bronchophony, and were usually most intense around the angle of the scapula, in the mid-scapular region, or at the top of the axilla.

5. **Adventitious Sounds.**—These were very various. In most cases might be heard loud, coarse, superficial gurgling râles, very much intensified over the base, and the areas where bronchial breathing was present. In other cases, again, the bronchial breathing was unattended with any adventitious sounds, whilst sometimes, cases were met with, where the breath sounds were weak, attended or not by râles; or occasionally, the breath sounds might be altogether absent.

Leathery frictions, creakings, wheezings, and sucking sounds, were also heard, and especially a peculiar sucking, creaking sound, difficult to describe, but not easily forgotten, not at all uncommon, and indeed seemed to us the only sign which of itself was at all peculiar to the condition.

The exact position of the disease in the lung is a question of no little importance, for, in ordinary cases of chronic tuberculosis of the lung, the disease is almost always most advanced in or near the apices. We did not find such to be the case in this series of cases. In the large majority, the disease was most intense and advanced at the base of the lung, and from the physical signs, one would be led to infer that the cavities or dilatations of the tubes, were most commonly found, just below the inferior angle of the scapula. Another point of distinction is that, when the disease passed over to the other lung, it was in every case the base of that lung which became affected. This order of things does not obtain in chronic tuberculosis; there, it is the apex of the opposite lung which becomes almost invariably affected.

In conclusion, it must be remembered that these cases are drawn from the poorest class of patients, and are, after all, not very numerous. These deductions must not be taken, therefore, as absolute facts, inasmuch as we feel certain that from the larger number of cases, and especially from cases in private practice, drawn from quite a different class, and living under entirely different circumstances, the conclusions might, in many instances, have been very different.

TABLES OF CASES OF FIBROID DISEASE COMPLICATED BY TUBERCLE.

No. of Case	Initials.	Sex.	Age.	Duration in Years.	Side.	Family History.	Personal History.	Appearance of Chest.	Heart.	Signs and Symptoms.	Tubercle Bacilli.	Temp.	Urine.	Pulse.	Fingers.	Remarks.
1	T. S.	M	47	12	R.	None of tubercle or fibrosis.	Good health until 12 years ago; then "congestion of lung" (right); slight cough and dyspnœa since; much worse 2 years ago.	Deficient movement and expansion over the right side; white spots in the skin.	Seen beating to right of sternum inside and beneath right nipple. No bruit.	For last 2 years cough. Expectoration (fœtid). Dyspnœa. Emaciation to extent of 18lbs. Slight hæmoptysis. He looks ill and wasted. Sweating. Percussion note impaired at apex and quite dull at base. Breath sounds cavernous all over, very little moist sound. A few crepitations in the left axilla.	In fair numbers.	Sub-febrile. Skin hot.	No albumen.	Quick.	Not clubbed.	Looks very ill, weak, and wasted.
2	E. B.	F	22	14	R. & L.	Mother's father had phthisis; none of fibrosis.	Measles at 7; since then cough; at 15 hæmoptysis; since then emaciation and sweating.	Thin, scanty covering; contraction and impairment of movement; no white spots.	Displaced; impulse in fourth interspace right nipple. No bruit.	Cough excessive, dyspnœa, expectoration, emaciation and sweating. All these have become worse since hæmoptysis at 15. Note impaired at apex and base; at latter position quite dull. Fine crepitations at apex, becoming coarse as base is reached. Bronchial breathing and bronchophony. (Fine crepitations at left apex.)	In large numbers.	Febrile.	No albumen.	95	Very clubbed, have been so 6 years.	Very frail and tuberculous-looking, thin and wasted.

No. of Case.	Initials.	Sex.	Age.	Duration in Years.	Side.	Family History.	Personal History.	Appearance of Chest.	Heart.	Signs and Symptoms.	Tubercle Bacilli.	Temp.	Urine.	Pulse.	Fingers.	Remarks.
3	T. S.	M	48	8	R. & L.	One sister died of phthisis; one child died of phthisis; wife died of phthisis; none of fibrosis.	Good health until 8 years ago; then cough, expectoration, and dyspnœa.	Thinly covered; contraction of right side; diminution of movement at both apices; many white spots.	Displaced to right; exact position not determined. No bruit.	Cough, expectoration, dyspnœa, wasting and night sweats, worse lately. Impaired note, dulness at base. Very loud gurgling râles at mid-scapular region, with amphoric breath sounds. Crepitations at left apex also.	None. One examination	Normal.	Albumen $\frac{1}{10}$th.	72	Slightly clubbed.	Since died; apparently tubercular.
4	E. J.	F	54	20	L.	Father died of dropsy at 77; one uncle and one aunt died of phthisis; one sister died of phthisis; husband died of phthisis.	Strong and well until 20 years ago, when husband died; then slight cough and wasting; no other illness.	Great contraction all over left side; hardly any movement; no white spots.	Not displaced. No bruit.	Cough, expectoration, wasting, streaky hæmoptysis. No diarrhœa. no sweating. Dulness from apex to base. Weak breath sounds. Scanty crepitations. Bronchial breathing at places. At base scarcely any breath sounds to be heard at all.	In fair numbers.	Sub-febrile.	No albumen.	96	Not clubbed.	
5	M. A.	F	51	3	L. & R.	None of tubercle or fibrosis.	Three years ago pain in left side and cough; no previous illness.	Contraction over left side; impairment of movement.	Displaced $\frac{1}{2}$ inch outside nipple line. No bruit.	Cough, expectoration, dyspnœa, wasting. streaky hæmoptysis, sweating. No diarrhœa. Impairment of note from apex to base. Well-marked signs of excavation at apex and base. At right apex impairment of note and fine crepitations.	Plentiful.	Sub-febrile.	A trace of albumen.	104	Not clubbed.	Certainly tubercular.

6	J. B.	M	50	11	L.	Father died of phthisis; one brother died of phthisis; none of fibrosis.	Had broncho-pneumonia in 1880; cough and expectoration since.	Thinly covered, contracted; impairment of movement; no white spots.	Displaced 1 inch outside nipple line: no murmur.	Cough, expectoration, wasting, sweating and dyspnœa. Attacks of profuse hæmoptysis. Impairment of note down left side. Crepitation, bronchial breathing at inferior angle of scapula. Fine crepitations at apex; right lung free.	Plentiful.	Sub-febrile.	No albumen.	Not observed	Markedly clubbed.	Tubercular.
7	F. L.	M	44	3	R.	One brother and one sister died of "inflammation of lungs"; no fibrosis.	Pneumonia 3 years ago; no previous illness; ill since the pneumonia.	Contraction and impairment of movement at right apex; no white spots.	Displaced; impulse in epigastrium; no murmur.	Cough, expectoration, and dyspnœa. Expectoration horribly fœtid. Hæmoptysis. Wasting 1½ stone in 1½ years. Pain. No diarrhœa. Dulness from apex to base in front. At *apex* in front evidence of a large cavity. No crepitations. Left lung free.	0	Normal.	No albumen.	0	Clubbed.	
8	H. D.	M	6	5	L.	None of tubercle or fibrosis.	Measles and whooping-cough at 1 year; cough ever since; no other illness	Impairment of movement on left side; no white spots.	Displaced ¼ inch outside nipple line. No bruit.	Cough, no expectoration. Progressive emaciation, sweating. No diarrhœa. No hæmoptysis. Dulness over left side. Evidence of a cavity at apex. No moist sounds. Breath sounds weak. Right side free. Liver and spleen enlarged, ascites. "Tabes mesenterica."	Sputum unobtainable.	Febrile.	Not examined	Quick.	Clubbed.	Tubercular.
9	C. B.	M	38	15	R.	Mother died of phthisis; brother died of phthisis.	Pleurisy 15 years ago, right side; cough and expectoration ever since; rheumatic fever 10 years ago.	Impairment of movement over the right apex; good at the base.	Displaced to right; mitral stenotic murmur.	Cough, expectoration, dyspnœa, frequent hæmoptysis. Emaciation to a slight extent, and slight night sweats. Impairment of note at right apex, clear at base. Crepitations. Breath sounds hollow at the base.	In fair numbers.	Not observed.	Not observed	Not observed	Not observed	Tubercular.

No. of Case.	Initials.	Sex.	Age.	Duration in Years.	Side.	Family History.	Personal History.	Appearance of Chest.	Heart.	Signs and Symptoms.	Tubercle Bacilli.	Temp.	Urine.	Pulse.	Fingers.	Remarks.
10	S. S.	F	19	6 months.	R. & L.	None of tubercle or fibrosis.	Contracted a pleurisy in left side 6 months previously, and has coughed since.	Distinct flattening over right side, and deficient movement and expansion.	Pulled over to the right side; no murmur.	Cough, expectoration, dyspnœa, slight attacks of hæmoptysis. Considerable emaciation; no night sweats. Impairment of note over *right side*. Breath sounds bronchial, attended with mucous râles, crepitations and creakings. Left side, crepitations and impairment of note at left apex.	In large numbers.	Raised.	No albumen.	Quick and frequent.	Not clubbed.	Looks tubercular.
11	S. T.	M	57	7	R.	None of tubercle or fibrosis.	Had "inflammation of the lungs" 7 years ago, and has had cough on and off ever since; is a stonemason.	Marked shrinking and deficiency of movement on right side.	Evidently displaced to the right. No bruit.	Cough, expectoration, and dyspnœa. Streaks of hæmoptysis. Has lost 3 stone in 6 years. No night sweats. Dull note all over right side. Bronchial breathing with bronchophony. Crepitations and creaking.	In fair numbers.	Normal.	No albumen.	74, strong	Not clubbed.	
12	W. D.	M	23	6	L. & R.	Mother died of phthisis; mother's family phthisical; none of fibrosis.	Well until 6 years ago, when he began to cough; there was no illness to account for the onset.	Left side extremely flattened; little or no movement there; movement also deficient over right apex.	Pulled upwards and outwards; apex in fourth space 1 inch outside nipple. No bruit.	Troublesome cough. Slight expectoration. Considerable emaciation. Night sweats. Hæmoptysis, two pints on one occasion Left side dull all over. Breath sounds bronchial, with crepitations, creaking and pectoriloquy. Evidence of infiltration & excavations at rht. apex.	In fair numbers.	Normal.	A trace of albumen.	96, small.	Clubbed.	

13	E. S.	F	23	10 months.	L. & R.	One brother died of phthisis; one sister died of phthisis; no fibrosis.	Ten months ago caught cold; cough ever since no illness to account for it.	Left side distinctly flattened and contracted; diminution of movement.	Not displaced; pulmonary second sound accentuated.	Cough, expectoration, dyspnœa. No hæmoptysis. Considerable emaciation. Night sweats. Impairment of note over lower part of left chest. Breath sounds attended with coarse bubbling râles. No distinct bronchial breathing. Crepitations and impairment of note at the right apex.	Large numbers.	100°–102°.	No albumen.	86	Slightly clubbed.

ANALYSIS OF THE CASES COMPLICATED WITH TUBERCLE.

We now turn to a consideration of the Tables of fibro-tubercular cases which are appended, and using the same methods as in the preceding pages, we will draw therefrom, such inferences as a small number of cases will allow. It will be useless to submit these Tables, which consist of 13 cases only, to the same minute inquiry, as we did in the fibroid Tables. For ideas and facts, gathered from such a basis, would inevitably lead to erroneous conclusions. However, we state our results, remembering that what was said at the beginning of this chapter, applies still more strongly here.

Of the 13 cases collected, 8 were males, and 5 females. The average age was about 37 years, the highest age being 57, and the lowest 6. In all cases of fibro-tubercular disease, as a rule, the average age was much greater than in pure fibroid disease, which it will be remembered was about 20 years.

Points of Interest.—The following points are of interest:

The Duration of the disease, as gathered from the 13 cases, was found to be about 8 years. This result is not in excess of the extreme duration of chronic phthisis, as set forth by Drs. C. J. B. and C. T. Williams, who found it, from an analysis of 1000 cases, to be about 8 years. On the other hand, it is considerably below the average duration of cases of fibroid disease, pure and simple, which, it will be remembered, was 11 years approximately. An investigation into the family antecedents did not throw much important light on the subject. In 7 cases there was a clear family history of tubercle, and in one case a doubtful history of fibrosis. In both the fibroid and fibro-tubercular cases, the tubercular predisposition was not more frequent than in a similar series of normal cases.

On turning to the **personal history** of the cases, we found evidence bearing upon the onset of the disease as follows:

The disease dating from an attack of—	
Measles and whooping-cough	2 cases
Pneumonia	4 „
Pleurisy	2 „
No definite onset	5 „

In connection with these figures, it may be said that, often no

reason at all for the onset of the disease can be determined. The patients begin coughing and wasting gradually, and this state of ill-health goes on indefinitely. Far different is this from the almost universal occurrence of broncho-pneumonia, or croupous pneumonia as the beginning of pure fibroid disease. This phenomenon is of importance from a diagnostic point of view, and will often help towards settling as to whether the process is of tubercular origin, or not.

We found **displacement of the heart** to be of frequent occurrence, it being present in all but 2 cases. In most cases the displacement was as much as in pure fibroid disease. We could discover no bruits in any of the 13 cases. The size of the heart, so far as could be estimated, seemed to be smaller than that with which we were already familiar in pure fibroid cases. The heart-beat also, differed materially from that of fibroid disease. Instead of the slow, measured, powerful, and infrequent contraction, which was almost pathognomonic of that affection, we found in this series of cases, the heart quick, frequent, and feeble ; in other words, we were dealing with a heart belonging to a patient with active tuberculosis of the lungs. Perhaps, of all the various phenomena in connection with the disease, this is the most striking, and the most constant. So much is this so, that an opinion, as to the true nature of the complaint, could almost with certainty be given, from a consideration of the heart alone.

The Pulse, too, gave evidence of a very different set of conditions. Instead of being slow, infrequent, and deliberate, the beats per minute ranged from 85 to 104. They were feeble, quick, and frequent. In fact, the pulse was nothing more than can be observed, any day, in an ordinary case of tuberculosis of the lungs, in which the disease is active.

Another important point of difference was the presence of **tubercle bacilli**. These were found sometimes in enormous numbers, sometimes only in small quantities, in 10 out of the 13 cases. They were found whenever looked for. In one case the expectoration was unobtainable, and in 2 cases only did we fail to examine for the presence of the microbe. Again, the temperature showed variations which had little in common with pure fibroid disease. In this series of cases it was raised in 8 out of the 13 cases, in some the evening peaks reached 102°, while in others, it was simply raised to about 99·5° or 100°. In 4 cases

it was normal while under observation; and in one case we were unable to verify it, owing to the patient not remaining a sufficient time under treatment.

It will be seen from the foregoing how very dissimilar are these two classes, the pure fibroid and the fibro-tubercular. The whole of this great change is brought about by the advent of bacillary phthisis upon a fibroid lung. But for this occurrence, we should have had no alteration in the heart, the temperature, and the pulse; the duration, instead of being curtailed, would have been as long as the other cases.

Frequency.—The tubercular variety of fibroid disease of lung is not so common as the non-tubercular, for in the three years during which we have collected, we have only met with 13 cases of the former, as against 45 of the latter variety.

Seat of Disease.—In 9 out of the 13 cases, the whole lung was more or less affected, and in many the apex more so than the base. In 2 cases the apex alone was affected, the opposite lung being free, whilst in the 2 remaining cases, the base of one lung was the seat of disease; but in both these cases, the apex of the opposite lung also showed signs of more recent trouble. In all the 7 double cases, on the side least and therefore probably last affected, the disease was of the apex. From this it will be gathered, that when tubercular, this disease tends sooner or later to affect both sides, and takes as its seat the apex rather than the base, therein differing largely from the non-tubercular form, in which one lung only is usually affected, and that most commonly at the base.

Albuminuria.—A trace of albumen was noticed in 3 out of the 13 cases, never large, and often disappearing. In 3 cases the urine was not tested.

Clubbing of Fingers was observed in 8 out of the 13 cases. In 2 cases it was very marked, whilst in some of the others it was only slightly in evidence.

It will be noticed, therefore, that with regard to the presence or absence of albumen, and the clubbing of the fingers, the two varieties show but little difference, these two signs apparently depending more upon the duration of the disease, and the amount of lung-tissue involved, than upon the fact that the disease was, or was not tubercular.

Symptoms.—In addition to those common to both varieties—viz., cough, dyspnœa, and expectoration—other symptoms, pointing still

more to tuberculisation of lung, showed themselves. Thus, hectic, wasting, and sweating were present, more or less marked, in most cases, making a striking contrast with the non-tubercular variety; as also did the character of the pulse, being weak, quick and soft in most, and never presenting the full, quiet, measured character, which was so striking a feature in the non-tubercular type. Moreover, the progress of the cases from bad to worse was infinitely more rapid and general.

Hæmoptysis.—This sign appeared with far greater frequency than in the non-tubercular variety, being present in 10 out of the 13 cases, and in most was considerable in amount.

The aspect of the sufferer was that of one afflicted with a rapidly progressive disease; indeed the patients were, almost without exception, weak, lacking in energy, unable to work, wasted and hectic in appearance.

Diarrhœa was not observed to be a marked feature in any of these cases.

Physical Signs.—As far as the lungs were concerned, these were naturally much the same in both varieties, bearing in mind that the seat of disease, as before mentioned, was much more often at the *apex* than at the *base*, and more frequently of *both* than of *one* side. The character of the heart-beat, too, in this variety, resembled the pulse in its quickness, weakness, and want of tone, as unlike that of the non-tubercular cases as the sounds of a heart could well be.

Displacement of Organs.—This was found very much on an equality in the two varieties; it must be noticed that with regard to the cardiac displacement, disease of both sides in this variety did not so often produce it, as in the other form, because the affection of the one lung was slight, recent, and always apical; conditions under which contraction has much less effect on the position of the heart.

CHAPTER VII.

A CASE OF FIBROID PHTHISIS.

[It has been thought well to add the following paper, which was read by Sir Andrew Clark before the Clinical Society on Feb. 14, 1868. It is reproduced here by the kind permission of the Council of the Society.]

Mary Sutherland, aged 28, married four years but childless, was admitted into the London Hospital, under the care of Dr. Andrew Clark, on November 21, 1867.

Patient states that she enjoyed good health till three years ago, when she was rather suddenly seized with ascites, for which she was treated in the London Hospital by Dr. Fraser; that she recovered in about eleven months; that she remained in tolerable health and certainly without any definite complaint till the close of July in the present year, when she was attacked with frequent vomiting followed by rapid loss of flesh and strength, harassing cough, attacks of hæmoptysis, muco-purulent expectoration, sleeplessness, and occasional diarrhœa; and that these symptoms have continued with greater or less severity to the time of admission.

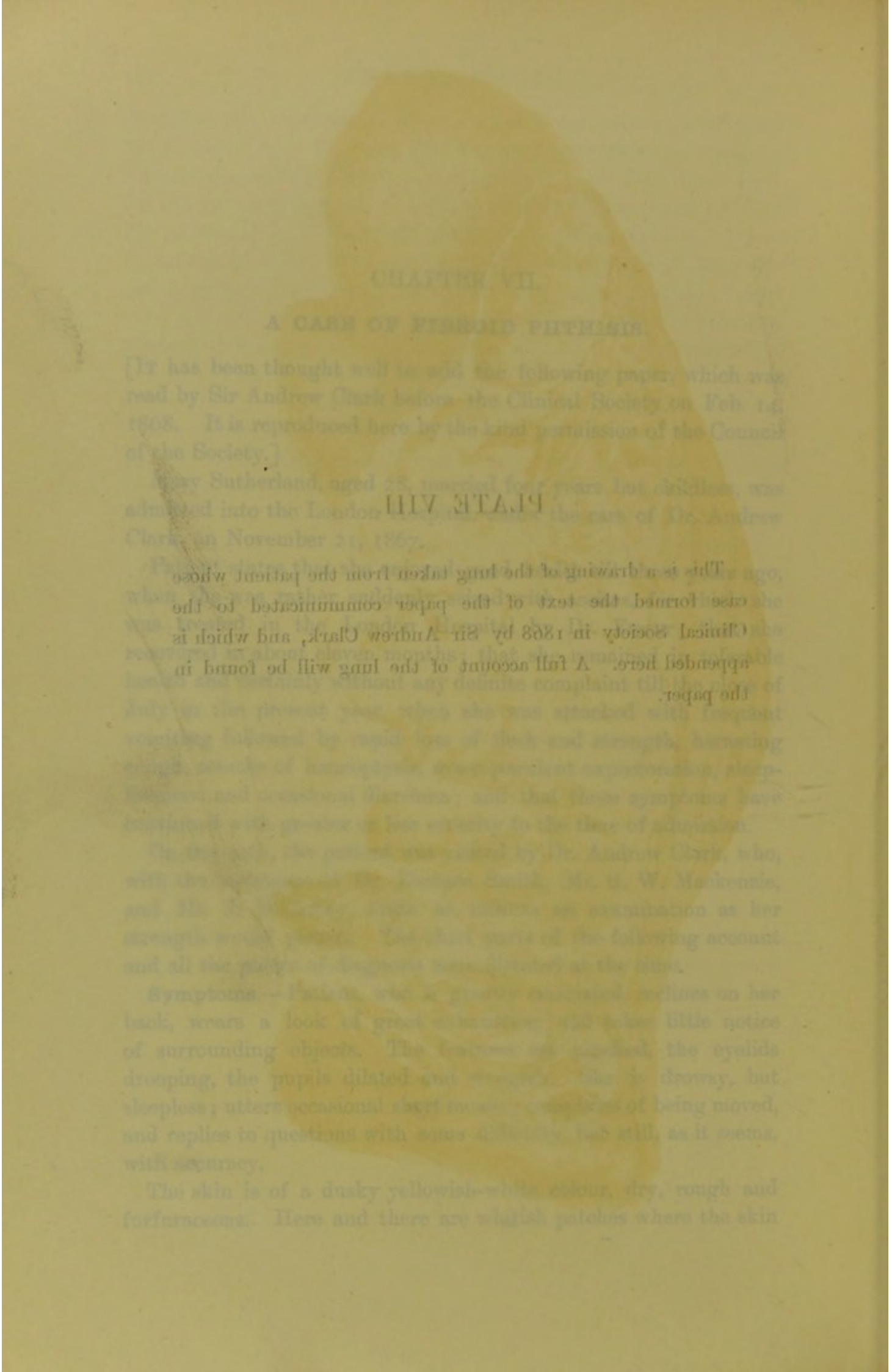
On the 24th, the patient was visited by Dr. Andrew Clark, who, with the assistance of Dr. Eustace Smith, Mr. G. W. Mackenzie, and Mr. J. M'Carthy, made as minute an examination as her strength would permit. The chief parts of the following account and all the points of diagnosis were dictated at the time.

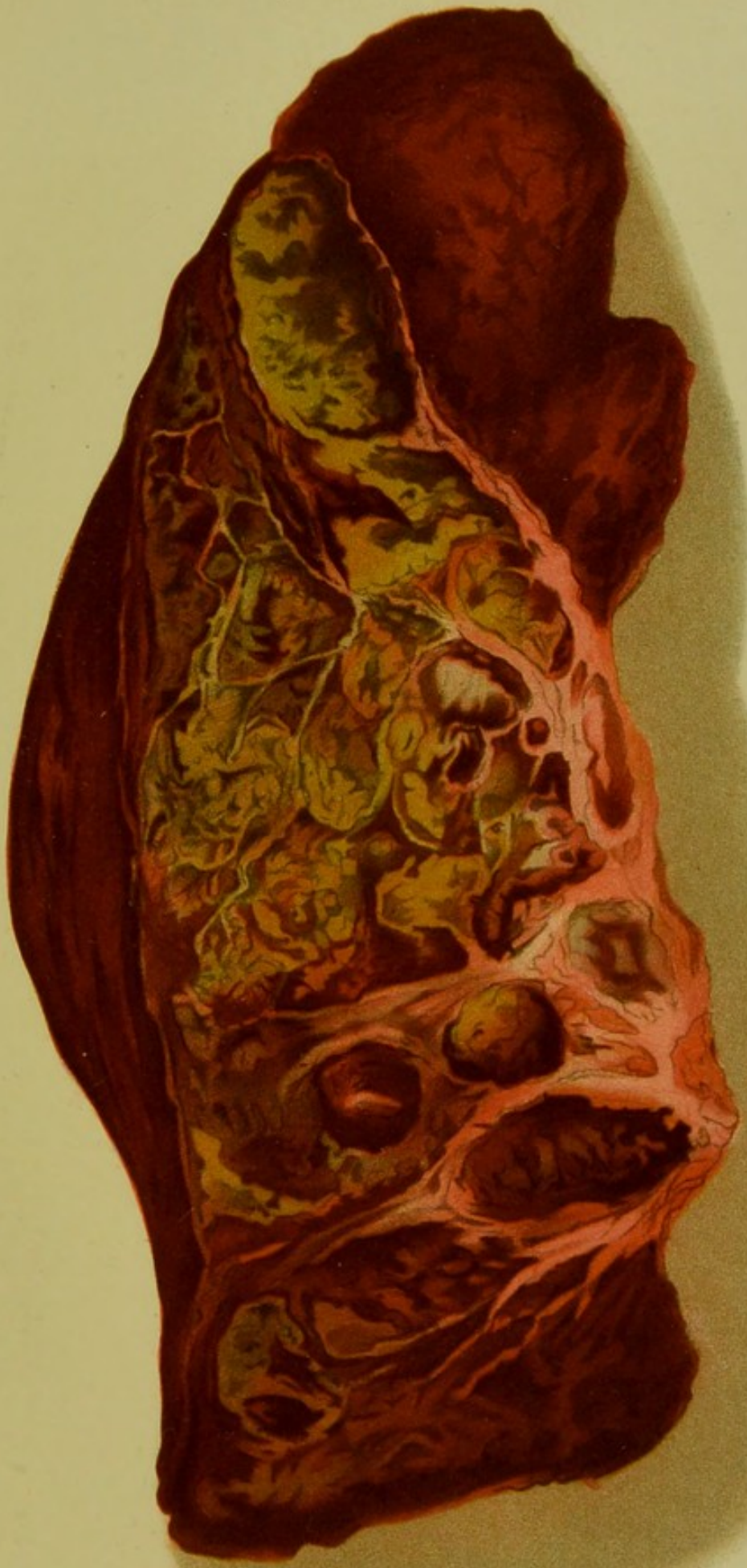
Symptoms.—Patient, who is greatly emaciated, reclines on her back, wears a look of great exhaustion, and takes little notice of surrounding objects. The features are pinched, the eyelids drooping, the pupils dilated and sluggish. She is drowsy, but sleepless; utters occasional short moans; complains of being moved, and replies to questions with some difficulty, but still, as it seems, with accuracy.

The skin is of a dusky yellowish-white colour, dry, rough and furfuraceous. Here and there are whitish patches where the skin

PLATE VIII.

This is a drawing of the lung taken from the patient whose case formed the text of the paper communicated to the Clinical Society in 1868 by Sir Andrew Clark, and which is appended here. A full account of the lung will be found in the paper.





seems denser and thinner than elsewhere. The hair is scanty, dark and coarse. The fingers are clubbed. In the axilla the temperature is 97·4° F.

Lower teeth almost all gone. Upper teeth and lips covered with sordes. Tongue denuded of epithelium, red, dry, cracked, shining, and protruded with difficulty. Urgent thirst. Frequent vomiting, aggravated by the introduction of food. Much belching of wind. Pain around the umbilicus increased by pressure. Abdomen yields no evidence of the presence of fluid. Bowels relaxed and relaxation preceded by griping pains. The hepatic dulness in the line of the right nipple extends two and a half inches below the free margins of the ribs; from thence the line of dulness runs obliquely upwards into the cardiac region where it is incapable of definition.

Urine, forty-five ounces in last twenty-four hours, pale, acid, and of the specific gravity of 1014. It contains a tenth of its bulk of albumen and deposits on standing a whitish cloudy sediment. By microscopic examination this is seen to consist of scaly epithelium, spheroidal epithelium, pus globules chiefly in masses, blood discs, and considerable quantities of granular matter. A second examination revealed the presence of a very few small hyaline tube casts. Patient has slight leucorrhœa. She menstruated naturally two months ago.

On account of patient's weakness only the front and lateral parts of chest were examined.

Respirations twenty in the minute, and calm though chiefly abdominal.

Examination of Left Side.—From second to fifth rib, and from the sternum to the front of axillary region, the chest wall is unevenly depressed and moves but slightly on inspiration. In the supra-clavicular region percussion is somewhat tympanitic; the breath-sounds are blowing; vocal resonance is bronchophonic and occasionally doubled as from echo. From the second to the fifth rib and from the left border of the sternum outwards and downwards into the axillary region there is marked dulness increasing in hardness and resistance from before backwards. In the fore part of this region the inspiratory sounds are of a blowing bronchial character and accompanied by moist subcrepitant râles; expiration, however, is not sensibly prolonged and not accompanied by crepitation. In the axillary part of this dull region the breath-

sounds are distinctly tubular and sniffling, though in some degree masked by moist crackling and coarse crepitation. Vocal resonance is for the most part bronchophonic and broken. At two spots, however, closely adjacent about the middle of the axillary region, the breathing is cavernous and the voice pectoriloquous. Here, too, a double metallic click is heard simultaneously with the heart's contractions. Throughout the outer and lower half of this dull space moreover there is heard at times a faint creaking leather sound, and the vocal fremitus is notably diminished. Over all the inferior part of this lung the inspiratory murmur is harsh, and the expiration, which in its first half is blowing, terminates only after apparent cessation of movements of thoracic walls in a few gentle puffs. Here, too, an exquisitely fine, dry, crumpling crepitation is developed in the course of forced inspiration. Vocal resonance and fremitus are both diminished.* Percussion over the anterior third of right lung tympanitic; elsewhere normal. Inspiratory murmur in front harsh and divided; expiration everywhere prolonged and accompanied by sibilant rhonchi. Vocal vibration increased. Occasional crepitation is heard over the root of the lung behind; but from a variety of reasons it is supposed to be conducted. Patient has no trouble in breathing. Cough, though not frequent, occurs in violent and fruitless paroxysms which end in vomiting. Expectoration occurs after a succession of fits of cough and then rather suddenly and in considerable quantity at a time. It seems muco-purulent and not lumpy or discrete. It is most abundant and sometimes fœtid in the morning, but does not exceed half a pint in the twenty-four hours. Microscopic examination reveals the presence of broken bands and areolæ of elastic tissue (disintegration of bronchial and alveolar walls). The chief constituents are pus globules with which are associated granule and pigment cells, blood discs, and much granular detritus.

Limits of cardiac dulness cannot be accurately defined. Apex of heart is seen and felt to beat against the upper border of fourth rib, a little outside and below the nipple, which is unusually high. In the upper part of second intercostal space, better than an inch and a half from the left border of the sternum, there is visible a pulsatile movement apparently synchronous with the ventricular contraction. At apex heart's sounds though feeble are normal.

* Ultimately in this region there arose uniform dulness, tubular breathing, and bronchophony.

At cartilage of second left rib a low pitched systolic murmur is heard which can be followed a little way upwards and outwards, but not downwards nor to the right side. It is variously modified by alterations of position and increased in pitch by a deep inspiration. Pulse 74, small and compressible. In a few places are signs of arterial degenerations.

Patient has no headache. Occasionally she experiences sensations of numbness or faintness in the limbs followed by tremblings. Hearing and sight somewhat defective: a very few white spots in the choroid. Lies with attention introverted, making frequent moans. When questioned she answers intelligently, though not very intelligibly, and complains of cough, thirst, vomiting, recurring abdominal spasms and great exhaustion. Is very drowsy, but has not slept well for several nights.

On concluding the examination, Dr. Andrew Clark expressed his conviction that the case was one of a constitutional character which had lately localised itself in an especial manner in the left lung; that the product of the constitutional disease was of a fibroid nature, and that it was probably due to syphilis or alcohol, or both together. Although the cough, hæmoptysis, diarrhœa, and the various physical signs of lung deposit above noticed, suggested the idea of an engrafted pulmonary tuberculosis, for which indeed the patient was admitted, Dr. Clark declared his belief that no such disease existed; that the case was not one of tubercular scrofulous or pneumonic phthisis, or of pulmonary cancer; but that it was one of fibroid phthisis, complicated with enlargement of liver, granular kidneys, ulcerations of the bowels, and fibroid degenerations of various organs and tissues.

Patient was ordered to have milk with ice and beef-tea jelly alternately in small quantities at short intervals; to have, instead of wine ordered on admission, small quantities of brandy, and to take an effervescing citrate of ammonia draught with two grains of iodide of potassium every four hours. A blister was applied to the epigastrium, and afterwards moist heat round the whole body.

Nov. 25.—Patient passed a restless night, feels no better, but seems weaker.

Mouth rather moister. Vomits frequently. Bowels confined. Pulse 78. Resp. 22. M. T. 94·4° F.; E. T. 98·2° F. In other respects the same.

Hot poultices were applied round the chest. Hydrocyanic acid

and morphia replaced the citrate of ammonia mixture. Brandy was ordered to be taken in soda-water.

27.—Weaker since last report. Tongue moister, takes a little food and retains some of it. Bowels still confined, and abdomen painful. Urine abundant and acid; sp. gr. 1018; one-tenth bulk of albumen; a few hyaline casts. Cough very troublesome. Little sleep. Pulse 80, feeble. Resp. 23. Skin moist at parts, M. T. 98·8° F.; E. T. 97·4° F. Feet œdematous.

To have fifteen grains of compound jalap powder at night, and to use the linctus glycerinæ for the relief of cough.

29.—Tongue moist, but red and fissured; less thirst and vomiting. Bowels relieved. Urine less abundant. Cough troublesome at intervals. Expectoration less. Had a restless night, but patient nevertheless expresses herself better. Pulse 84, very small. Resp. 24. M. T. 100·1° F.; E. T. 98·2° F.

Dec. 1.—Much worse since last report. Tongue dry; great thirst; recurrence of vomiting. Bowels relaxed. Urine less abundant, alkaline and more albuminous. Paroxysmal attacks of cough without expectoration. Has painful sensations of breathlessness, chest oppression, and sinking. Cold sweats about the hands, feet, and face. Œdema extending. Great restlessness. Pulse 90, small and irregular. Resp. 26. M. T. 99·2° F.; E. T. 97° F.

Half an ounce of brandy mixture every hour. Chlorate of potash lemonade. ℞ Spts. chloroformi ♏xv, spts. am. ar. ♏xx, liq. morphiæ hyd. ♏vijss, aq. camph. ℥j, q. quarta hora.

2.—Had a better night through means of half a grain of opium. Is obviously, however, much weaker. Takes no food. Vomiting has ceased. No movement of bowel. Urine alkaline and albuminous. Cough less: loud moist râles over both lungs in front. Breathing calm, but somewhat irregular. Complains of oppression of chest. Skin clammy. Patient takes little notice, but still answers correctly when questioned. Pulse 92. Resp. 20. E. T. 96·2° F.

3.—Patient became comatose during the night, and died this forenoon.

Post-mortem Examination.—Dec. 4, at 4 P.M., the general examination was made by Dr. Hughlings-Jackson, and the microscopic one by Dr. Andrew Clark; but for the following account of both the latter is alone responsible.

Head not examined.

Thorax.—Heart small and drawn upwards to the left side. Pericardium adherent to left lung, which at the point where it overlapped the origin of the vessels, was indurated. Cardiac valves and orifices healthy. Right lung of large volume; anterior and inferior margins pale, dry, and emphysematous. Bronchial mucous membrane, where examined, thick, soft, red, and coated with a viscid bloody mucus. Interlobular areolar tissue everywhere healthy. No deposit in any part of lung. Left lung universally and firmly adherent to the pericardium, ribs, and diaphragm. When removed it was seen that upon the surface corresponding to the first and last of these parts the adventitious lymph formed a thick white dense membrane, which sent septa inwards through the lung. Over the surface corresponding to the ribs the lymph was thinner, softer, speckled with capillary blood extravasations, and evidently of recent origin. The lung was irregular in form and diminished in bulk. It measured barely six and a half inches from apex to base. Taken into the hand it felt about its middle like a hard, dense, but elastic fibrous growth. A section having been made through the lung from summit to base, the following appearances were presented: From the root of the lung, which was embedded in dense fibroid tissue, whitish ramifying septa shot outwards to the circumference traversing every part of the lung but the apex, which, except at one point where there was a sub-pleural nodule,* was quite healthy. In addition to these septa, which followed the course of the bronchi and blood-vessels, there were other septa intersecting and occupying the place of the interlobular tissue, and organically connected with the thickened pleura. The portions of lung lying between these fibrous bands were variously altered. Commonly the imprisoned lung was hard, dense, resistant, and of a greenish slate colour. At certain other parts the lung exhibited rounded cheesy lumps, a few as small as peas, the majority as large as hazel-nuts. The smaller ones occupied the centres of lobules, and the larger ones were for the most part accurately limited by the interlobular tissue. Some of the latter had broken up into small rounded cavities. From the root of the lung there passed diagonally outwards to the circumference a chain of oval communicating cavities each rather larger than a Spanish nut. So free was the communication, that the whole chain looked like one

* This nodule was merely a mass of fibrous tissue about the size of a pea.

elongated cavity partially intersected by shallow partitions. These cavities were lined with cheesy matter and contained some curdy pus streaked with blood. On close examination it seemed as if the cavity nearest the root of the lung had originally arisen from a dilated bronchus. Three rather thick septa traversed the lower third of the lung horizontally. Between the first and second were several small rounded communicating cavities; between the second and third the lung was simply grey, solid, and tough. At the pleural extremity of the third was a cavity about the size and shape of a very small walnut. Below the third septum and at the anterior free margin of the organ were numerous cheesy deposits accurately filling the lobules, which were all isolated and distinctly mapped out by apparently unaffected interlobular tissue.*

The walls of the pulmonary artery were distinctly thickened as far as they could be followed with the naked eye, and the thickening was plainly due to some change in the outer coat of the blood-vessel. All the horizontal septa were formed by tough, white, pervious or impervious blood-vessels. The walls of the bronchial tubes were also thickened, and here and there slightly dilated; but there were no such marked and general dilatations as are sometimes met with in common cirrhosis. Some of the bronchi terminated in seeming continuity with cavities. The mucous membrane was generally thick and vascular: at a few places in the larger tubes it was ulcerated and villous. There was everywhere marked prominence of the longitudinal elastic bands and transverse muscles. No grey granulation was anywhere to be found in this lung. Bronchial glands enlarged: two of them contained cheesy deposits.

Abdomen.—The liver weighed 6 lbs. 7 ozs. Capsule healthy. Parenchyma pale, dense, very friable, and exhibiting all the appearances of waxy (amyloid) degeneration. The surface of a section, which had a faintly nutmeg appearance, exhibited reddish-brown spots on being brushed with the iodine solution, but no violet colour was developed by it and sulphuric acid together. Bile pale, watery, and abundant. Spleen weighed 11½ ozs., and was also waxy. Capsule thickened by layers of old lymph. Both kidneys small, granular, and contracted. The granulations, however,

* This had exactly the appearance, as may still be seen in the preparation in my possession, of what is called cheesy, scrofulous, and by me epithelial pneumonia.—A. C.

were larger and paler than is common in the ordinary cirrhotic kidney. When laid open, the cut surfaces had a faintly waxen appearance, and developed a few reddish-brown spots on the application of iodine. Numerous deposits and ulcerations in the lower part of the ileum. The deposits were seated in the submucous tissue, more or less spherical, and in size varied from the magnitude of a pin's-head to that of a pea. The larger ones when pricked gave out a semi-fluid matter like curdy pus. Some of them had ruptured naturally, and exhibited the appearance of minute excavated ulcers with thickened margins. The other ulcers were oval, and lay across the long axis of the gut. They had granular everted borders, shallow vascular bases, studded with minute cup-like depressions. The peritoneum over them was intensely vascular, and coated with films of soft lymph. Here and there over the ulcers were thin circular patches, looking exactly as if drops of melted wax had fallen upon the membrane and then solidified. Mesenteric glands enlarged, but apparently not otherwise altered.

Summary of Microscopic Examination.—Bronchial glands. Close examination of sections of the non-cheesy glands revealed the presence of minute semi-transparent sclerous spots or tracts. When portions of these parts were submitted to microscopic examination, and compared with other portions of parts apparently healthy, the only recognisable difference between them was the presence in the former of an amorphous substance, which, by its accurate adaptation to and limitation by the alveoli, suggested the notion that it had been produced in a liquid form and had afterwards solidified. At all events, the most careful analysis revealed no new structural form which might possibly have been considered as the agent of its production. At one or two points where the sclerous substance occupied several adjacent alveoli, there was the faintest appearance of fibrillation, but none of distinct fibres. The cheesy matter occupying one or two of the glands consisted of granular matter, free nuclei, and disintegrating cells. The gland substance surrounding this matter was full of large cells, obviously multiplying by nuclear proliferation and extrusion.

Lungs.—The finer horizontal septa were found to consist of obliterated blood-vessels or bronchi, the structural elements of which had undergone various degrees of retrogressive metamorphosis. In these, as well as in the still pervious vessels and tubes,

the areolar tissue coat was more or less thickened. The intersecting fibrous bands occupying the interlobular fissures as well as the bands shooting inwards from the thickened pleura, consisted of a true areolar tissue, which appeared to have grown by a process of differentiation, and not at all by any mere process of cell development. The elastic elements appeared to have sprung from the nuclei, and the inelastic, or white, either from the direct solidification of a fluid exudation, or from the differentiation of already existing fibres. Certainly no other spindle-shaped or fusiform cells were found except those which obviously came from the walls of the bronchi or blood-vessels.

The portions of lung more immediately embraced by these septa were in one of four conditions. They were either emphysematous, or congested, or had a dense slate-coloured fibroid aspect, or contained cheesy deposits.

The emphysematous portions of lung were simply atrophied. The alveoli contained no cells; there was no trace of spherical or oval nuclei, no indication of blood-vessel, and the alveolar wall appeared to consist only of a homogeneous riddled membrane, traversed by a few fibres. The blood-vessels leading to such portions were invariably found to be obliterated.

In the congested parts the alveoli contained numerous epithelial and pus-like cells, with some granular matter and blood discs.

In the dense slate-coloured fibroid-looking parts the alveoli were accurately filled by what seemed at first sight to be a perfectly amorphous substance, having only here and there the slightest appearance of fibrillation. And in many cases no manipulation and no reagent brought to light any structural form in the midst of this amorphous substance. Nevertheless, in the majority of cases, dilute solutions of acetic acid or of potash revealed the presence of epithelial or pus-like cells in one or several regular or irregular layers lying against the alveolar walls. But it was worthy of notice that these cell-forms were only such as were found where there was no sclerosis and only slight congestion.

It now became evident from repeated comparative examinations that three distinct morbid processes had been going on in the lungs: one, the spread of a true areolar tissue from the pleura, the interlobular fissures, the bronchi, and the blood-vessels; the second, the production of a fluid which infiltrated the textures and clotted into amorphous moulds of the alveoli; and the third, a simple

fibroid withering or degeneration of the normal textures. Wherever true fibrous tissue was found in any part of the lung substance, it could be traced back to blood-vessel, or bronchus, or interlobular fissure. Though the alveoli therefore were invaded from without, there was no independent production of fibrous tissue within them; the sclerous change which some of them exhibited was due to the presence of an amorphous substance arising in a different manner, and only occasionally exhibiting traces of fibrillation. Lastly, it was noticed that wherever the sclerous change was most complete, the circular nuclei, which in health stud the alveolar walls, were either broken up into granules, or altogether absent.

The smaller cheesy deposits occupying the centres of lobubes were composed of flattened epithelial-like cells with a few blood discs, particles like the white corpuscles of the blood, and some granular matter. The larger masses, involving whole lobules and extending over several adjacent ones, had a somewhat different constitution, owing probably to greater age or quicker development. Speaking in general terms, the centre parts of each consisted almost entirely of a granular detritus. Around this ran a zone of débris studded with nuclear-like particles and cellular shreds. Still more externally, lay heaps of disintegrating nucleated cells. The circumference was crowded with similar bodies, swollen and variously distorted—some having many nuclei, others numerous vacuoles—all evidently in a condition of active proliferation and decay. Scarcely a granule cell was to be seen, and but very little free fat. Of course the structural elements were not arranged in such precise order at all parts as may appear from this description. The description is, nevertheless, drawn from nature and substantially accurate. To the naked eye the consolidations just described had exactly the characters of what is still described in, unhappily, the majority of our schools and books as "cheesy tubercular infiltration." But the microscope showed very plainly that they possessed the exact structural characters of what the author has called epithelial or scrofulous, and the chief foreign schools, quite incorrectly, a cheesy pneumonia. Quite incorrectly, because it is now beyond question that both the leading types of pneumonic exudation, the simple acute (corpusculo-fibrinous, according to the author)* and the catarrhal or cheesy (epithelial in the writer's arrangement), are alike liable in certain conditions to undergo the cheesy metamor-

* Lectures delivered at the Royal College of Physicians, 1866.

phosis. To designate one pathological condition by a quality possessed, though less frequently manifested, by another, is a plain and unallowable violation of the proprieties of a scientific terminology.

If the word tubercle is to signify anything definite in opposition to everything indefinite—if it is to be employed in a rigorous method as an instrument of progress in opposition to the loose and shifty manner of the schoolmen in their controversies about prevenient grace, as a mere means of successful logomachy, and thereby the cause of endless perplexity and confusion—the consolidations above described cannot be called tubercular. They are the results either of a vesicular and lobular epithelial (cheesy) pneumonia or of a simple scrofulous process akin to that which, independently of obvious inflammation, issues in the stuffing of an organ or texture with large spheroidal, nucleated, quickly growing, and prematurely decaying cells. Happily the main object of the present communication renders it unnecessary to determine to which of these pathological processes the consolidations in question are to be referred. Moreover, such a discussion could be successfully conducted only upon an occasion when the point at issue formed the chief subject, and not an incidental topic of interest.

Liver.—In sections of the liver it was seen that the textures were pervaded by some amorphous substance, and that the nuclei of the capillary walls were greatly multiplied, rather smaller than normal, and spindle-shaped instead of oval. One territory of liver cells was pale, nebulous, and waxy; another dark, granular, and fatty. The former became reddish-brown, the latter light yellow on the application of iodine. In several trials the vessels remained quite unaffected by this reagent. The ordinary arrangement of cells in a lobule was broken through. Instead of being disposed in cylindrical rays diverging from a centre, and connected by transverse bars, the cells, in the waxy parts, were huddled together in apparently confused and shapeless heaps.

Kidneys.—A homogeneous, and here and there fibroid substance, much in excess of what any system of shrivelled capillaries could form, surrounded the tubes in the cortex. The capillaries were studded with oat-shaped nuclei. The tubes here contracted and there dilated, or almost cystiform, contained at some points granular matter, and at others what seemed to be hyaline cylinders. Some of the Malpighian capsules were accurately filled by a nebulous or waxy

substance, which suggested the idea of its origin by means of the exudation of a fluid which had suddenly solidified. Scattered spots throughout the whole organ became reddish-brown on the application of iodine; but between the parts that were, and those that were not, affected by this reagent, no structural difference could be detected.

* * * * * * *

Remarks.—It cannot be doubted that of late years considerable advances have been made in most departments of the science and art of medicine. But in one—in that which relates to the pathology and treatment of lung diseases—it does seem that, notwithstanding much movement, there has been little progress. For, if we proceed to consider the character of the change born of often-recurring and yet seldom-varying conflicts of opinion, by comparing the knowledge of these days with the knowledge current in the times of Baglivi and Avenbrugger, the judgment naturally growing out of the comparison is that the change, certainly not in most things for the better, is probably in the main for the worse.

There are two chief reasons for this—the one pathological, the other clinical.

The pathological reason is that men at the outset of their inquiries adopting the provisional formula of Laennec get infatuated by its simplicity and persist in declaring it to be absolute. "Every cheesy lump is a tubercle. Tubercle is a new growth resulting always from a special constitutional vice, and never from a neglected cold—an unabsorbed pneumonic deposit or a fibroid invasion of the lung. Pulmonary phthisis is simply the suppurative disintegration of the tubercular deposit. However different may seem the anatomical changes effecting and accompanying the destruction of the lung in this disease—grey lumps or yellow—circumscribed or diffused—acute or chronic—fibrous or tubercular—pneumonic or hæmorrhagic—they are all, but varieties the one of the other. The modes of expression may be many; the essence is but one. For other organs there are, it is admitted, various agents of destruction: for the lung, however, there is practically none but tubercle."

Much that is plausible can be urged in support of these views; and it requires rare freedom from traditionary influences, much careful observation, and the closest thought to become completely convinced of their fallacy. When, however, we learn that the

cheesy state is but a common stage in the downward history of the most diverse things—not of tubercle alone, but of mucus and pus and lymph and blood—we are bound to reject as false the dogma that every cheesy lump is a tubercle and every cheesy mass a tubercular infiltration. When, furthermore, we discover in the bodies of persons dying of what is conventionally called consumption, pathological states of lung of a constitutional as well as of a local character, different in their mode of origin, in their structure, disposition, effects, progress, and issues, we are justified in believing that phthisis is not one but manifold. It is true, in fact, as Bayle said speculatively of old: "Phthisis is a genus including several species." The generic term comprehends all progressive consolidations and circumscribed suppurative disintegrations of the lung: the specific term should indicate by a distinctive adjunct the different states concurring to this end. For surely if the progressive consolidation and suppurative destruction of lung constituting phthisis be determined in one instance by tubercles, in another by pneumonic exudations, in a third by scrofulous growths, in a fourth by fibrous invasions, and if these things be in any sense different from one another, common sense demands that their differences should be permanently recorded by distinctive designations. Hence it is both convenient and correct to speak of tubercular, scrofulous, pneumonic, fibrous, and bronchial phthisis.

There is also a **clinical reason** for the present unsatisfactory condition of our knowledge and treatment of pulmonary affections. The symptoms indicating their presence have a closer dependence upon mere damage to the function of the lung than upon the varying nature of the pathological process effecting it. There are therefore of necessity striking resemblances between the clinical characters of all lung diseases. But there are likewise radical differences; and if one's perceptions were not blunted by long familiarity and biassed by preconceived opinion, one might arrange these differences into natural groups and discover for each an independent centre of pathological change. That under the guise of pulmonary phthisis are hid diverse affections of the lungs is a truth now, as we have seen, beyond the reach of doubt; and so to set forth in a definite manner the assemblage of symptoms by which these affections are severally to be distinguished during life, has become the prime desideratum in the pathology of lung diseases. For until this is done, the results of clinical inquiry

must continue radically corrupt, the conclusions drawn from therapeutic experiment delusive, and our management of the phthisical state, at all times uncertain, often mischievous.

It is plain, then, from what has been said, that to employ the term tubercle in a "comprehensive sense"—that is, in a sense depriving it of any definite meaning—and to maintain, as so many distinguished persons still strive to do, the unity of phthisis; is to reject the most precious results of pathological research, to ignore the distinctions established by clinical inquiry, to relinquish the hope of a rational therapeutic, and to resign one's self for ever to the guidance of a blind and capricious empiricism.

The patient whose history is above recorded seems to have been the subject of constitutional fibroid degeneration (fibrosis), which, affecting lightly now one organ or texture and then another, at last localised itself in an especial and destructive manner in the left lung. It is therefore described as a case of "fibroid phthisis."* In this term the author proposes to embrace all those cases, whether local or constitutional, which are anatomically characterised by the presence, in a contracted and indurated lung traversed by more or less dilated bronchi, of fibroid tissue, and of a tough fibrogenous substance, together with cheesy deposits or consolidations, and usually small cavities commonly found about the middle and lower parts of the affected organ. Several objections may be justly made to the designation proposed; but in the writer's mind they are all outweighed by this great advantage: the name expresses the leading anatomical fact of the disease, and whilst theories of its nature will change, the structural character must remain always the same. The history of what was once called the inflammation globule, and exudation corpuscle is sufficient to convince one of the evil effects of a hypothetical naming. Even now pathology has not recovered from the perplexity and confusion introduced by this reprehensible terminology.

The case, though in several ways imperfect, has been selected for discussion because its main points are still fresh in the recollection of numerous persons who saw the patient during life; and because, though a bad example of the disease intended for illustra-

* This term is also used, though in a more restricted sense and with somewhat different views, by Laycock, perhaps the first to recognise the distinctive nature and importance of cases like the one now described, and by H. G. Sutton in his important and able paper on Fibroid Degeneration.

tion, it will be a good test of the strength of those arguments by which its claims to a distinctive name will be assailed.

At the bedside, even at the first examination, a diagnosis was not difficult to frame. Tubercle was excluded by the absence of any evidence of structural disease in the right lung. Had disease of such extent as existed in the left lung been tubercular, the right lung would not have been free from evidence of kindred lesions. Besides this, the left apex was presumed to be free; there was no great hurry of the circulation, no evening fever, no continued elevation of temperature, and no profuse perspirations. Until the advent of death, the skin was dry and inactive. Lastly, in the presence of positive signs of another constitutional disease, the occurrence of diarrhœa cast no serious doubt upon the propriety of the exclusion. The author is conscious that this opinion is not shared by others. Sutton especially mentions the absence of ulceration and of diarrhœa, as points of diagnostic importance. But the present case affords another illustration of the fact, which experience has peremptorily taught the writer, that the occurrence of ulceration of the bowels in the course of chronic disease of the lungs is not conclusive as to its tubercular character. Deposits in, and ulcerations of, the intestinal glands may occur in almost any form of chronic disease to which the lung is liable. The diagnosis lay between chronic fibrinous pleurisy, common cirrhosis, and cancer: there was no other disease which could account for the contraction of the lung and the displacement of the heart. But the contraction did not occupy the place, and was plainly not of the composite character peculiar to contractile pleurisy. Moreover, disintegration of lung was proved, if not by the physical signs, at least by the presence of lung-tissue in the sputum. This last reason also sufficed to show that the disease of the lung was not a mere cirrhosis. Was it, then, a cancer? The aspect of the patient, the rapid progress of the disease, its unilateral character, the retraction of the chest wall, the continuity and extent of the dulness, and the frequent attacks of hæmoptysis, all disposed one to the adoption of this conclusion. But then the very considerable contraction of the lung accompanied by moist râles, and a justifiable conviction of the presence of several scattered cavities, the absence of any evidences of outgrowth, enlarged glands or tumour in other parts, and the presence of positive signs of constitutional fibroid degeneration, eliminated the idea of cancer, and left to one

the only tenable conclusion that the case under consideration was one of fibroid phthisis.

The existence of cheesy deposits in the lung was inferred partly from mere pathological experience, and partly from a belief in the existence of ulcerations in the bowels. Cheesy matter—that is, almost any pathological product in the way of retrogressive metamorphosis*—appears from experiments, often repeated by the author, to be in some manner an efficient cause of secondary deposits and diarrhœa.

The systolic basic bruit was inferred to be dynamic, and dependent on pressure by reason of its variability, and the readiness with which it was influenced by changes of position, inspiration, and coughing. Some such bruit is nearly always present in advanced fibroid disease of the left lung.

The other points in the diagnosis admit of no further comment at present.

These remarks may, with propriety, be closed by a summary of the more important circumstances which can help one to understand the nature and correctly diagnose the presence of fibroid phthisis.

1. Fibroid phthisis may have a constitutional or a local origin (Laycock; C. J. B. Williams; Handfield-Jones).

2. When the disease has a constitutional origin, the fact will be indicated by the presence of signs of fibroid disease in other organs and tissues; by white skin spots (H. G. Sutton); by corneal degenerations (Canton); by anæmic urine containing granular matter, granular casts, or albumen; by evidences of cirrhotic or amyloid liver, of enlargement and hardening of spleen, and of induration of the nervous centres (Laycock).

3. The constitutional state of which these growths, deposits, or degenerations are the manifestations seems to be closely connected with, if not dependent upon, the abuse of alcohol (Huss), syphilis (Wilks), rheumatism (Laycock), gout (author), exhausting discharges (Dickinson), and defective excretion (author).

4. The actual product of this state appears either as a true fibroid tissue, or as a tough, hard, amorphous, fibrogenous sub-

* That is so long as structural disintegration is incomplete. When all structural forms are destroyed, and the cheesy mass is composed of nothing but molecular débris, it loses in great measure, if not entirely, its power of producing secondary deposits.

stance (Williams; Walshe). The latter is closely akin to the "amyloid material," and even in the lung is sometimes reddened by iodine. It does not appear to be the product of cell proliferation, but to be the result of a real exudation in the ordinary sense of that term. The former can always be traced into continuity with perilobular, peribronchial, perivascular, or subpleural areolar tissue. The latter occupies the alveoli, and is often found in isolated patches. Structural elements, such as are seen in adjacent parts, are often found in it. When nuclei are present in it, they are the ordinary circular nuclei of the alveolar walls, and not the spindle-shaped or oat-like nuclei of connective tissue. In other organs, the fibrogenous material sometimes contains oat-shaped nuclei derived from proliferation of the vascular walls. To this source also are to be referred many of the nuclear fibres and spindle-shaped cells, supposed to be illustrations of the cellular development of fibrous tissue.

5. When the disease is not of constitutional origin, there will be found evidence either of the pursuit of some occupation exposing the patient to the inhalation of irritating particles (Peacock; Greenhow), of the existence of tubercular phthisis, long-continued bronchitis, or of some former attack of acute disease, such as fibrinous pleurisy, pericarditis, or what the author has called the corpuscular form of pneumonia.

6. The disease commonly affects one lung, and more commonly the left than the right.

7. If the disease affects both lungs, it is, as a rule, either the effect of mechanical irritation, or it is accompanied and has probably been caused by the presence of tubercles.

In rare cases, the double affection may be caused by rheumatism or syphilis (Wilks).

8. In fibroid phthisis there is always contraction of the chest wall. The percussion dulness is harder, higher pitched, and more uniformly continuous than in any other lung disease. Resistance of thoracic parietes is greatly increased. Intercostal spaces are depressed. Sometimes the dulness is tubular. Vocal fremitus is at one time increased, at another greatly diminished. Over the fibroid lung one hears blowing breath-sounds, often without audible prolongation of expiration; occasionally coarse, dry, and moist râles, superficial creaking, and diffuse bronchophony. Over cheesy deposits of any extent the breath-sounds are sharply tubular, the

expiration prolonged, and the vocal resonance bronchophonic, sniffling, and circumscribed.

9. If the apex of the diseased lung is early involved in the solidification, there is reason to suspect the existence of tubercles ; if there is moist crackling in the supra-spinous fossa, the suspicion amounts to probability ; and if with the slightest dulness over the summit of the opposite lung there is any moist crepitation, doubt is practically no longer possible.

10. When the lung below the solidification is healthy, expiration is peculiarly prolonged and puffy.

11. When cavities exist, they commonly occupy the mammary region : occasionally they are found in the base, and with extreme rarity in the summit of the lung. (Sutton's cases appear to have been complicated with tubercles.)

12. When about the middle of the lung several cavities lie near together in a horizontal or diagonal line, and are bounded by solidified lung, they are in all probability due to dilated bronchi. Cavities resulting from the breaking up of cheesy deposits are commonly isolated, irregularly placed, larger, and yield on auscultation unmistakable cavernous or amphoric breathing and pectoriloquy. The only certain evidence, however, of the existence of cheesy cavities is the presence of areolæ of elastic tissue in the sputum.

13. In the neighbourhood of the solidified lung it is not uncommon to find patches and tracts of lung in a condition of extreme vesicular emphysema; and the atrophic changes constituting it appear in many instances to be due to plugging, or some other obliteration of branches of the pulmonary artery. It is therefore a statical as opposed to a dynamical emphysema.

14. When the left lung is affected, the heart is usually displaced upwards, and a little outwards. When the right lung is affected, the heart is drawn chiefly outwards and a little upwards. In both cases, a low-pitched systolic bruit is commonly heard over the pulmonary artery.

15. The cough is paroxysmal, and ordinarily induces vomiting. The expectoration varies : it is usually yellowish, greenish, or ashen grey, studded with pigment streaked with blood ; sometimes fœtid, and ejected with difficulty after several ineffectual fits of exhaustive coughing.

16. The general symptoms, viewed collectively, are strikingly

different from those of tubercular phthisis. The skin is, local sweats excepted, dry and inactive; there are no profound exhaustions, no continuous elevation of temperature, no evening fever, seldom any hectic flushing. Till the disease is far advanced, the breathing is quiet, and the pulse below 84.

17. Slight œdema of the lower extremities is very common in the course of this disease, and almost always present at its close.

18. Patients who have long suffered from fibroid phthisis, whether of local or constitutional origin, become at last pallid, waxy-looking, and cachectic.

19. The disease is commonly slow in its progress, and when it complicates tubercles it retards disintegration and greatly prolongs life. This may explain some of the cases of unusually protracted phthisis in spirit-drinkers.

20. The frequent occurrence of even copious hæmoptysis, and the setting in of diarrhœa in the course of this disease, may justify a suspicion of the development of tubercles, but are not conclusive evidences of their presence. Hæmoptysis, diarrhœa, and ulceration of the bowels often occur during life without a solitary tubercle being found after death.

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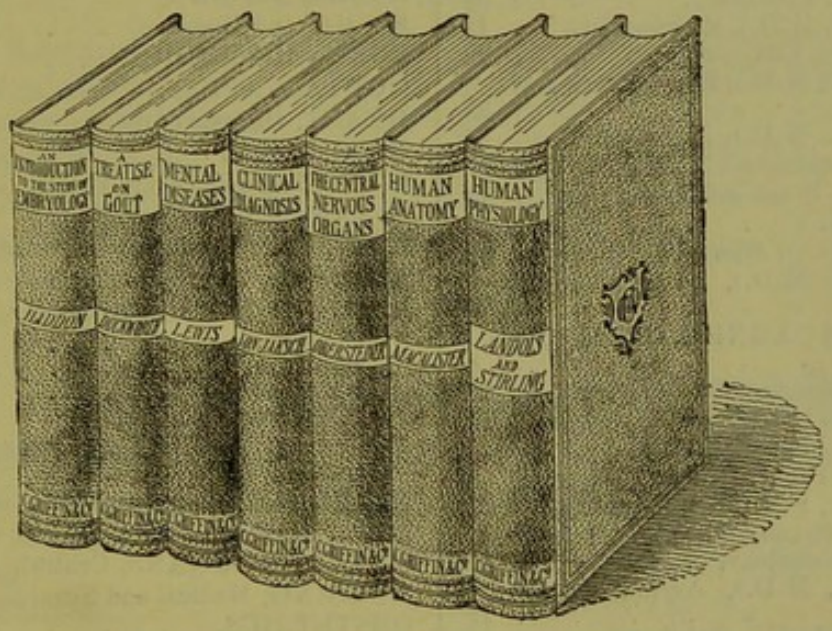

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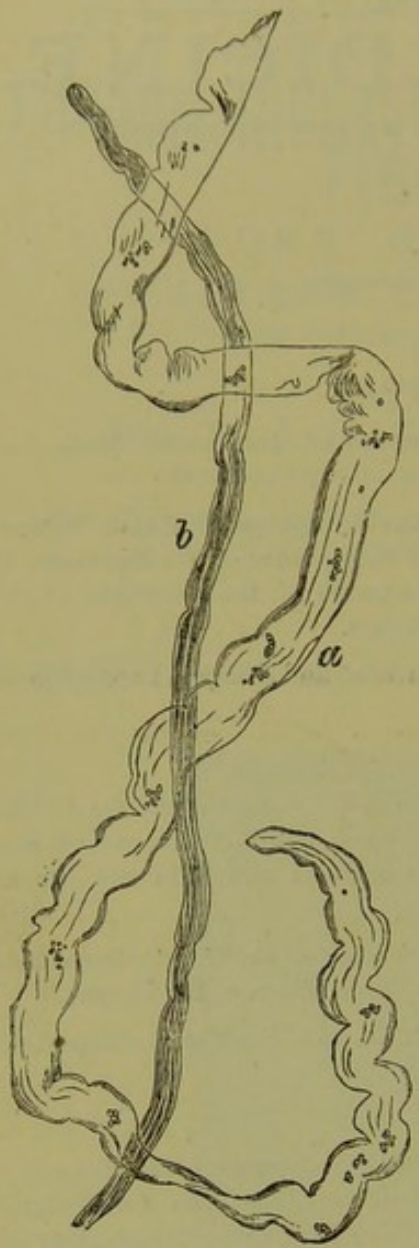

Fig. 86.—*a*, *b*. Cylindroids from the urine in congested kidney.

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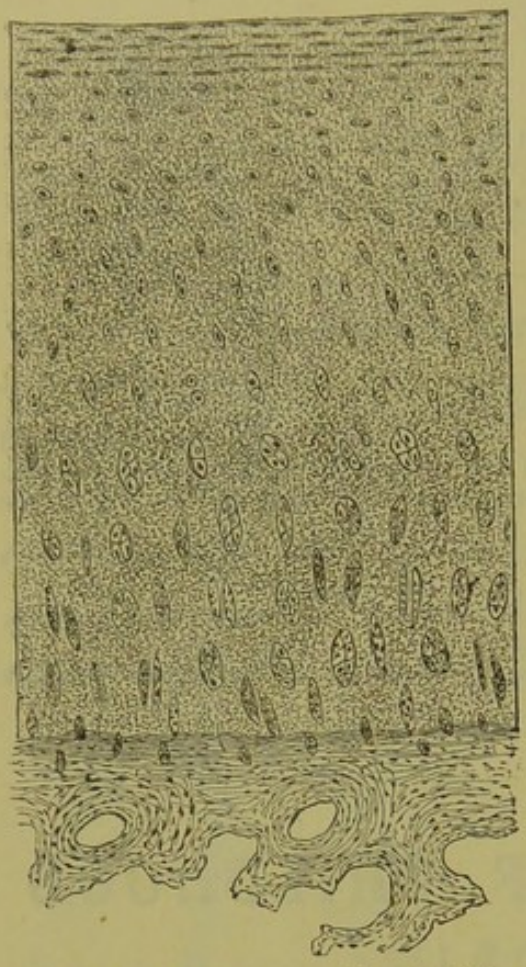

Fig. 1.—Human Articular Cartilage from head of a metatarsal bone (Normal).

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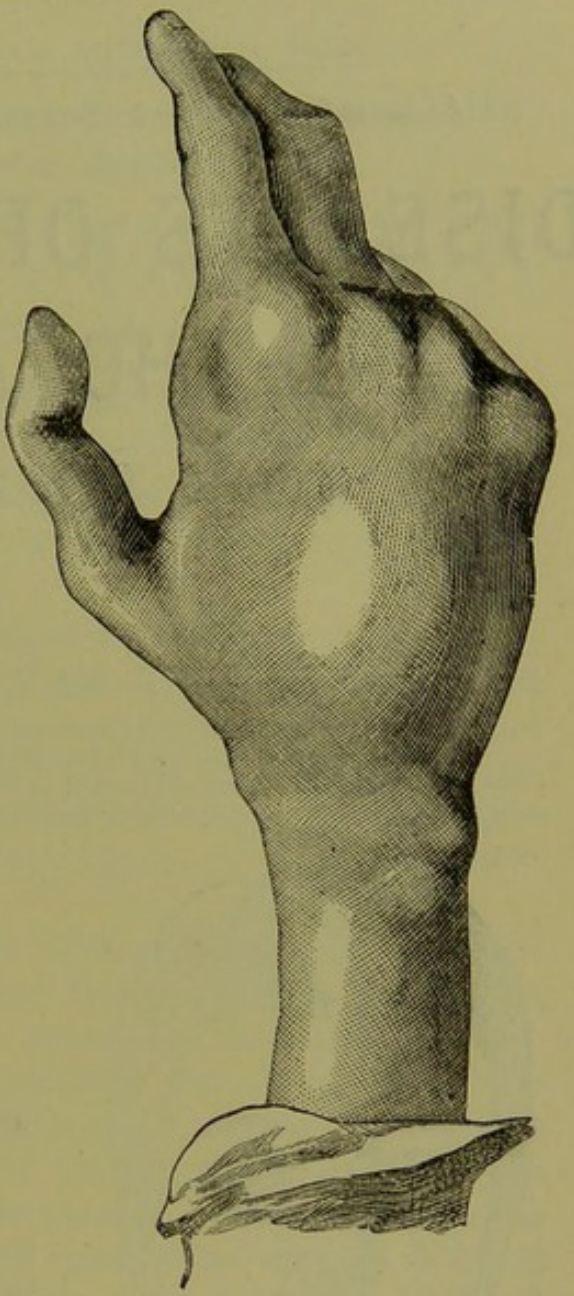

Fig. 1.—Gangliform Swelling on the Dorsum of the Hand of a Child aged Eight.

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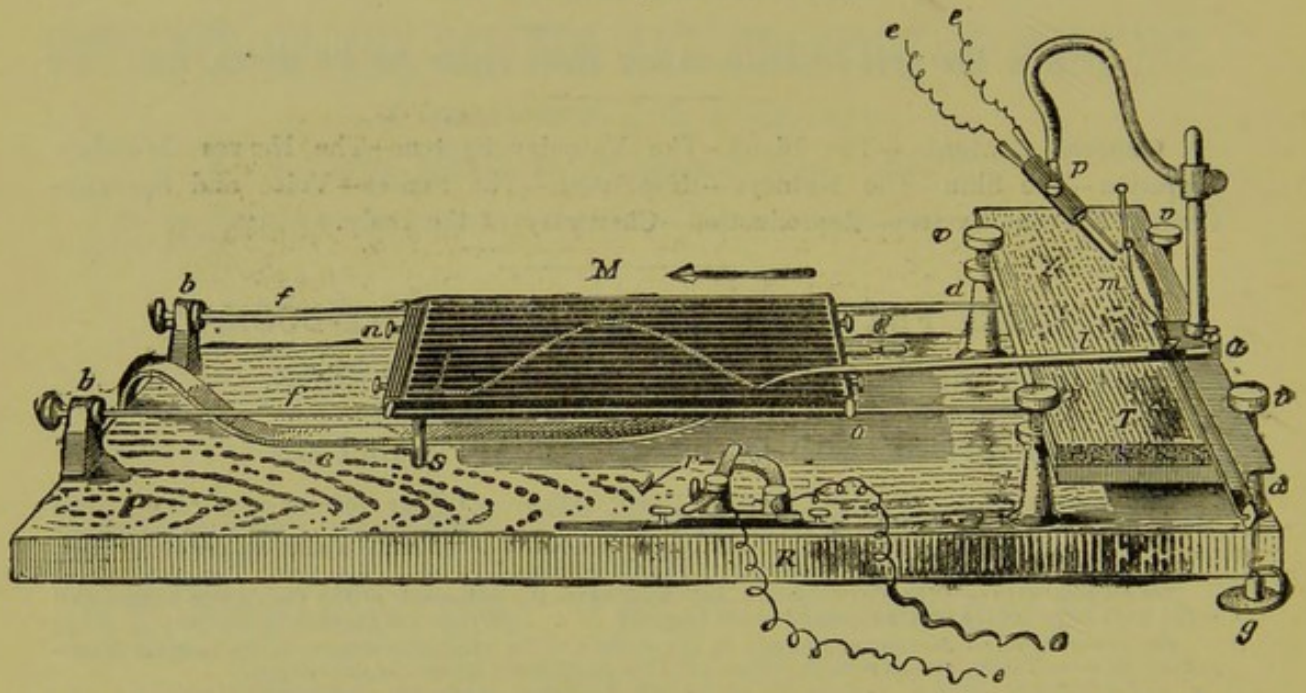

Fig. 118.—Horizontal Myograph of Frédéricq. *M*, Glass plate, moving on the guides *f, f*; *l*, Lever; *m*, Muscle; *p*, *e*, *e*, Electrodes; *T*, Cork plate; *a*, Counterpoise to lever; *R*, Key in primary circuit.

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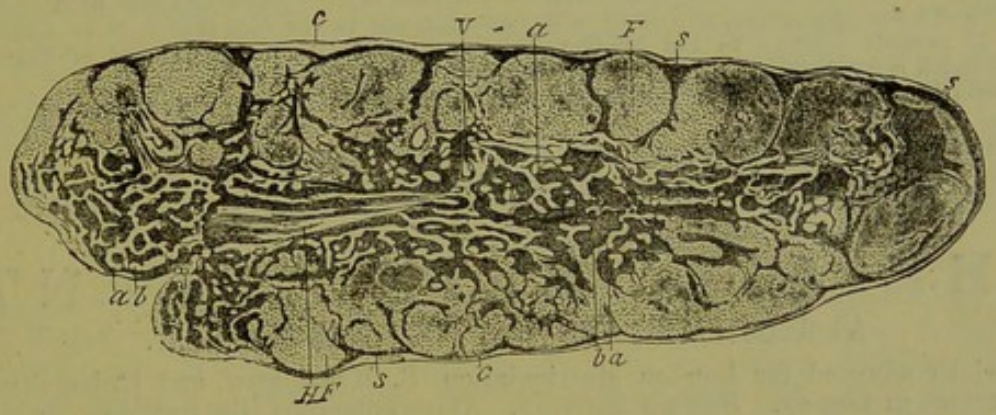

Fig. 200.—L.S., Cervical Ganglion of Dog. *c*, Capsule; *s*, Lymph sinus; *F*, Follicle; *a*, Medullary cord; *b*, Lymph paths of the medulla; *V*, Section of a blood-vessel; *HF*, Fibrous part of the hilum, × 10.

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